

Podrid's Real-World

ECGs

A Master's Approach
to the Art and Practice
of Clinical ECG Interpretation

Volume 5 **Narrow and Wide Complex
Tachyarrhythmias and Aberration** **Part A: Core Cases**

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Forewords by: Hein J.J. Wellens, MD • Roman W. DeSanctis, MD

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Volume 5 **Narrow and Wide Complex Tachyarrhythmias and Aberration—Part A: Core Cases**

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These workbooks are dedicated first to my wife Vivian and son Joshua, whose patience, tolerance, support, and love over the years have been limitless, exceptional, and inspirational. They are also dedicated to the many cardiology fellows, house staff, and medical students whom I have had the pleasure and honor of teaching over the past three decades and who have also taught me so very much.

Philip Podrid

To my wife Cindy, daughter Sapna, and son Sanjay, for all their love, support, and encouragement.

Rajeev Malhotra

To my darling daughters, Mia and Eila, whom I love to infinity.

Rahul Kakkar

For Katie and Jack

Peter A. Noseworthy

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Contents

Foreword

by Roman W. DeSanctis, MD ix

Foreword

by Hein J.J. Wellens, MD xi

Preface xiii

Introduction

Narrow and Wide Complex Tachyarrhythmias and Aberration.... xv

Real-World ECGs: The Cases

Cases 1–36: Wide Complex

Tachyarrhythmias and Aberration—Core Cases 1

Cases 37–61: Narrow Complex

Tachyarrhythmias—Core Cases 204

Index 353

Additional cases for further study are available in a separate digital edition,

Volume 5, Narrow and Wide Complex Tachyarrhythmias and Aberration—

Part B: Practice Cases.

Foreword

The invention of the electrocardiogram (ECG) by Dr. Willem Einthoven, first reported in 1901, ranks as one of the all-time great discoveries in medicine. Einthoven's landmark achievement was duly recognized in 1924, when he was awarded the Nobel Prize in Medicine.

By the early 1940s, all of the components of the 12-lead ECG that we use today were in place. When I finished my cardiology training 50 years ago, the ECG was one of very few cardiognostic tools available to us. As a result, we received an intensity of training in electrocardiography that is generally not encountered in many of today's cardiology fellowship programs, where the emphasis has shifted toward the newer high-tech diagnostic modalities. Yet the ECG remains a major pillar in the evaluation of disorders of the heart. In a patient with a cardiac arrhythmia, what diagnostic information does the treating physician want the most? Of course—the ECG. Although the medical world progresses rapidly and changes constantly, the body of knowledge surrounding the ECG is virtually timeless. What was true 50 years ago is largely true today, and will remain so 50 years from now.

This wonderful series of ECG workbooks, appropriately entitled “Real-World ECGs,” by Dr. Philip Podrid and three outstanding young cardiologists from Massachusetts General Hospital—Dr. Rajeev Malhotra, Dr. Rahul Kakkar, and Dr. Peter Noseworthy—offers a splendid opportunity for self-education in electrocardiography (and a bit of fun at the same time). An esteemed academic cardiologist, Dr. Podrid has had a career-long interest in electrocardiography. Over many years he has collected and saved thousands of ECGs for teaching

purposes, and it is a portion of his incredible collection that has been used to spawn these books.

There are scores of textbooks on electrocardiography, but what sets these volumes apart is that every ECG is tied directly to an actual clinical case. Each ECG is initially presented in a visually attractive and readable format accompanied by a clinical vignette. On the next page, the salient features of the ECGs are highlighted, dissected, and discussed in meticulous detail, followed by a summary of the patient's clinical problem and treatment, particularly as they relate to the ECG findings.

The first volume in this unique series covers electrocardiography basics. It is followed by five more volumes covering the entire spectrum of electrocardiography: myocardial abnormalities, conduction abnormalities, arrhythmias, narrow and wide complex tachycardias, and a sixth volume amalgamating a potpourri of paced rhythms, congenital abnormalities, and electrolyte disturbances. As I perused one of the workbooks, I truly enjoyed the experience. It is fun to try to guess the clinical problem from the ECG. In fact, on my teaching rounds, that is often exactly what I do. I will ask the trainee to present first just the ECG and with other trainees try to deduce from it what might be going on clinically. For example, in an adult with marked left ventricular hypertrophy and strain, one of three conditions is almost always present: severe aortic valve disease, hypertrophic cardiomyopathy, or hypertensive heart disease.

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These books should prove to be valuable for the teaching and learning of electrocardiography at all levels—from nursing and medical students to residents to cardiology fellows to practicing internists and cardiologists. They should be especially helpful for those seeking board certification or recertification in cardiovascular diseases, where knowledge of electrocardiography still is given a very high priority.

There is one further important component for those who utilize this series. In addition to the six workbooks, hundreds of other ECGs handled in a similar format are available online. From clinical diagnoses to interactive questions to patient management, cardiotextpublishing.com offers ECG-centric clinical cases for the viewer to further master the art of ECG interpretation.

Anyone who reads these books and views the auxiliary electronic material cannot help but be impressed by the prodigious amount of work that went into their preparation. Drs. Podrid, Malhotra, Kakkar, and Noseworthy should be justifiably proud of the final results of their Herculean efforts. I am confident that other readers will find these books and their electronic supplement as informative and enjoyable as I did.

Roman W. DeSanctis, MD

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Massachusetts General Hospital

James and Evelyn Jenks and Paul Dudley White Professor of Medicine

Harvard Medical School

Foreword

The electrocardiogram (ECG) was born in the Netherlands at the beginning of the 20th century when physiologist Willem Einthoven made the first recording of the spread of electrical activity in the beating heart from the surface of the body in a living human being. Since then, the ECG has become the indispensable “workhorse” in the management of patients suspected to have a cardiac problem.

The reasons are obvious. An ECG can be obtained anywhere. A recording is easily and quickly made, noninvasive, inexpensive, reproducible, and patient-friendly. The ECG gives instantaneous diagnostic information, is essential in selecting appropriate management, and allows documentation of the effect of treatment in cases of acute and chronic cardiac ischemia, rhythm and conduction disturbances, structural changes in the cardiac chambers, electrolyte and metabolic disorders, medication effects, and monogenic ECG patterns indicating the likelihood of cardiac abnormalities. The ECG is also a valuable tool for epidemiologic studies and risk stratification of the cardiac patient.

In the 110 years during which the ECG has been in use, we have seen continual improvements in its value in light of information gleaned

from other invasive and noninvasive diagnostic techniques, such as coronary angiography, intracardiac localization of abnormal impulse formation and conduction disturbances, echocardiography, MRI, and genetic evaluation. This means that not only does the novice health care professional need to be informed about all the information currently available from the ECG, but the more senior physician also needs to stay up-to-date with ever-evolving new developments.

Dr. Philip Podrid is known worldwide as an expert in electrocardiography. He is also a superb teacher. When you combine his input with beautiful ECGs, not surprisingly, you will have a series of “Real-World ECGs” that demonstrate the art and practice of clinical ECG interpretation as only a real master can. I hope that many readers will profit from this exceptional educational exercise.

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Preface

The electrocardiogram (ECG) is one of the oldest technologies used in medicine and remains one of the most frequently obtained tests in the physician's office, outpatient clinic, emergency department, and hospital. ECGs continue to play an essential role in the diagnosis of many cardiac diseases and in the evaluation of symptoms believed to be of cardiac origin. The ECG is also important in the diagnosis of many noncardiac medical conditions.

Like any other skill in medicine, the art of ECG interpretation requires frequent review of the essentials of ECG analysis and continual practice in reading actual ECGs. However, many health care providers who wish to augment their expertise in the interpretation of ECGs and develop the skills necessary to understand the underlying mechanisms of ECG abnormalities have realized that the currently available resources do not adequately meet their needs.

Teaching in medical schools and house staff programs does not typically emphasize ECG analysis. Consequently, many physicians do not feel adequately trained in interpreting the ECG. The currently available textbooks used for teaching ECG analysis are based on pattern recognition and memorization rather than on understanding the fundamental electrophysiologic properties and clinical concepts that can be applied to an individual ECG tracing, regardless of its complexity. The physician is not, therefore, trained in the identification of important waveforms and subtle abnormalities.

The workbooks and website of *Podrid's Real-World ECGs* aim to fill the gap in ECG education. These unique teaching aids prepare students and health care providers of all levels for the spectrum of routine to challenging ECGs they will encounter in their own clinical practice by providing a broad and in-depth understanding of ECG analysis and diagnosis, including discussion of relevant electrophysiologic properties of the heart, associated case scenarios, and clinical management.

The Workbooks

Each of the six volumes in *Podrid's Real-World ECGs* teaches the art of ECG interpretation by careful analysis of specific examples and identification of important waveforms. Each ECG is taken from a real clinical case and incorporates a discussion of important diagnostic findings and essential associated electrophysiologic mechanisms, as well as critical clinical management decisions. The purpose of the series is to provide readers from all fields of medicine with a systematic approach to ECG interpretation using a concise, case-based format.

This volume, the fifth in the series, delves into narrow and wide complex tachyarrhythmias and aberration. The other volumes focus on

continues

the basic approaches to reading any ECG as well as on other disease entities for which the ECG is useful:

- Essential introduction to the basics of ECG reading, outlining the approaches and tools that are utilized in the interpretation of all ECGs (Volume 1)
- Atrial and ventricular hypertrophy, acute myocardial ischemia, acute and chronic myocardial infarction, and pericarditis (Volume 2)
- AV and intraventricular conduction disturbances and enhanced AV conduction (Volume 3)
- Rhythm analysis, covering sinus, atrial, junctional, and ventricular arrhythmias (Volume 4)
- Miscellaneous conditions, including pacemakers, electrolyte disorders, and acquired and congenital cardiac conditions (Volume 6)

Each volume in the series starts with a didactic introduction that addresses the important ECG findings associated with each clinical category. This is followed by core illustrative case-based ECGs that lead the reader through identification of the important ECG findings associated with the specific abnormalities being discussed and provide information about the basic electrophysiologic mechanisms involved. This section is followed by a random assortment of topic-related ECGs. Every ECG presents a clinical scenario to further enhance the student's

skills at ECG analysis. Importantly, each case presentation is followed by an in-depth discussion of the ECG findings, with the important waveforms on the ECG highlighted.

The Website: www.realworldECGs.com

In addition to the didactic ECG cases found in the workbooks, the website (www.realworldECGs.com) offers optional access to a large, searchable repository of supplementary case-based ECGs. This ancillary material offers further practice in ECG interpretation using interactive case studies with Q&A that includes feedback and discussion about the important findings and clinical issues involved.

The benefit of a Web-based program is that many more ECGs can be presented and ECGs demonstrating specific abnormalities can be accessed quickly. In addition, the ECGs can be read using an approach that is similar to how they are analyzed in clinical practice—by identifying the waveforms important for diagnosis. Each of the relevant features is highlighted independently, providing a useful way to approach ECG reading.

This versatile Web-based program allows the user either to interpret ECGs in random fashion or to focus attention on a specific topic or ECG finding. This approach allows ECG interpretation to be performed in a way that is most effective for the user.

Philip Podrid, MD

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Rahul Kakkar, MD

Peter A. Noseworthy, MD

Introduction

Narrow and Wide Complex Tachyarrhythmias and Aberration

Narrow Complex Tachyarrhythmias

Classification of Narrow Complex

(Supraventricular) Tachyarrhythmias (FIGURE 1)

1. Sinus node
 - a. Sinus tachycardia: physiologic (catecholamine-mediated)
 - b. Sinus tachycardia: non-physiologic
 - i. Inappropriate sinus tachycardia (hyperkinetic heart syndrome)
 - ii. Sinus node reentry
2. Atrium
 - a. Atrial tachycardia-unifocal
 - i. Multifocal atrial tachycardia (MAT) (rate >100 bpm)
 - ii. Wandering atrial pacemaker (WAP) or multifocal atrial rhythm (rate <100 bpm)
 - b. Atrial flutter
 - c. Atrial fibrillation
3. Atrioventricular junction/node
 - a. Ectopic junctional tachycardia
 - b. Atrioventricular nodal reentrant tachycardia (AVNRT)
 - c. Atrioventricular reentrant tachycardia (AVRT) occurs due to an accessory pathway (Wolff-Parkinson-White, Lown-Ganong-Levine)

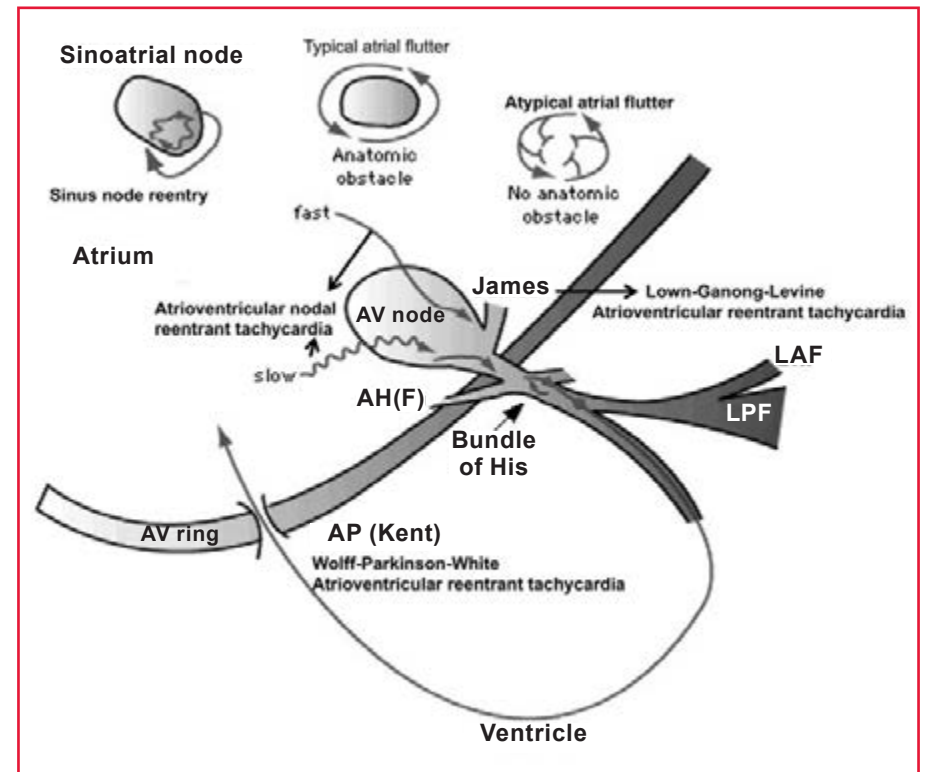


Figure 1. Anatomic pathways responsible for the arrhythmias involving the sinus node (sinus node reentry), atrium (typical and atypical atrial flutter), atrioventricular node (atrioventricular nodal reentrant tachycardia), and preexcitation (atrioventricular reentrant tachycardia) in Lown-Ganong-Levine or Wolff-Parkinson-White.

ECG Clues to Differentiate the Mechanism of a Supraventricular Tachyarrhythmia

Most important first step is “CHERCHEZ LE P”, *ie*, look for the P wave

1. P-wave morphology

- a. Sinus: The P wave is upright in leads I, II, aVF, and V4–V6. It is inverted in aVR. There is only one P-wave morphology.
- b. Atrial tachycardia (usually ectopic): There is only one P-wave morphology, but it is different from the sinus P wave, *ie*, it may be negative or biphasic (negative–positive) in leads where it should be positive. The PR interval may be longer or shorter than that seen with sinus rhythm. If sequential P waves are seen (as a result of AV block), then the baseline between P waves is isoelectric and flat.
- c. Multifocal atrial tachycardia (MAT) and wandering atrial pacemaker (WAP): There are ≥ 3 distinct P-wave morphologies (with no one P-wave morphology that is dominant) and variable PR intervals.
- d. Atrial flutter: There is regular and uniform atrial activity with a nonconstant (undulating) baseline between atrial waves. The intervals between the atrial waveforms are constant, and there is a uniform morphology and amplitude. In typical atrial flutter, the waves are usually negative–positive (sawtooth) in leads II, III, and aVF. In atypical flutter, the waveforms are usually positive in these leads.

- e. Atrial fibrillation: There is no organized atrial activity and hence no distinct P wave. Atrial activity may be seen, but it is irregular in morphology, amplitude, and interval.
- f. Ectopic junctional tachycardia/AVNRT/AVRT: There is no P wave seen before any QRS complex. There may or may not be a retrograde (negative) P wave (especially in aVF that is the lead perpendicular to the atria) after QRS complex. If there is a retrograde P wave seen, the RP interval is constant.

2. P wave or atrial rate

- a. Sinus tachycardia: 100–180 bpm
- b. Atrial tachycardia: 120–220 bpm
- c. Typical atrial flutter: 260–320 bpm
- d. Atypical atrial flutter: >320 bpm
- e. Ectopic junctional tachycardia: 100–200 bpm
- f. AVNRT: 120–220 bpm
- g. AVRT: 140–240 bpm
- h. Atrial fibrillation: >350 to >450 bpm
- i. MAT: 100–220 bpm, variable PP interval (irregularly irregular)
- j. WAP: <100 bpm, variable PP interval (irregularly irregular)

3. RP/PR relationship (FIGURE 2)

- a. No-RP tachycardia: No P wave is seen after the QRS complex, or it is at the very end of the QRS complex, superimposed on the terminal portion (RP interval <0.08 sec).
 - i. Typical AVNRT (called slow-fast) is the most common etiology.

- ii. Atrial tachycardia is a rare etiology.
- iii. AVRT is an uncommon etiology.
- b. Short-RP (long PR) tachycardia: There is a distinct P wave shortly after the QRS complex, often in the ST segment. The P wave is closer to the QRS complex before it than the QRS complex that follows it. The RP interval is constant.
 - i. AVRT is a common etiology. P wave is negative or retrograde in at least aVF.
 - ii. Typical AVNRT (often called slow-slow due to relatively slow conduction of the fast pathway) is an uncommon etiology. P wave is negative or retrograde in at least aVF.

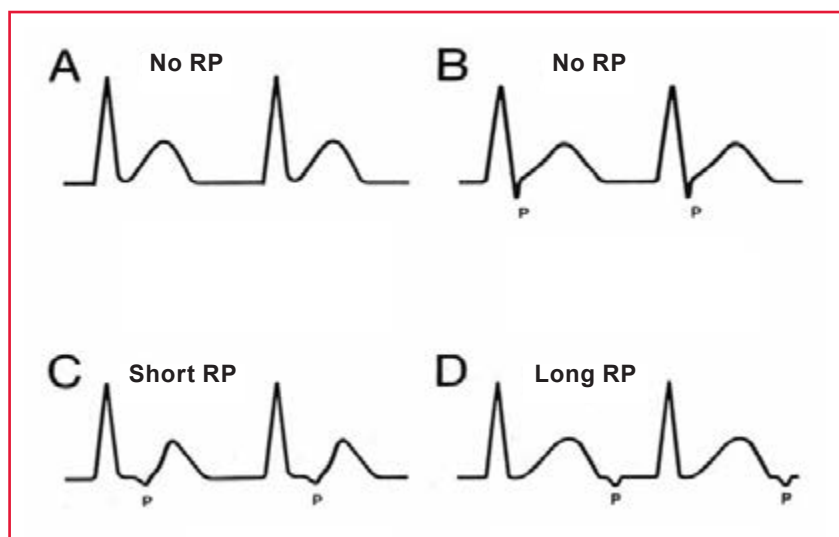


Figure 2. Waveforms indicating the relationship between the QRS complex and P waves in a no RP, short RP, and long RP tachycardia.

- iii. Atrial tachycardia is a common etiology. P wave is different than sinus P wave.
- iv. Atrial flutter (with 2:1 AV block) is a common etiology.
- v. Ectopic junctional tachycardia with retrograde atrial activity (P wave negative in at least aVF) is a common etiology.
- vi. Sinus tachycardia with first-degree AV block is a common etiology. P wave is normal in axis, *ie*, positive in leads I, II, aVF, and V4–V6.
- c. Long-RP (short PR) tachycardia: There is a distinct P wave that is closer to the QRS that follows it and further away from the QRS that precedes it. The RP interval is constant.
 - i. Typical AVNRT (called fast-slow) is an uncommon arrhythmia. P wave is retrograde and negative in at least aVF
 - ii. AVRT is an uncommon etiology. P wave is retrograde and negative in at least aVF
 - iii. Atrial tachycardia is a common etiology. P wave is different than sinus P wave
 - iv. Sinus tachycardia is a common etiology. P wave has normal axis, *ie*, positive in leads I, II, aVF, and V4–V6.
 - v. Atrial flutter (with 2:1 AV block) is a common etiology.
 - vi. Ectopic junctional tachycardia is an uncommon etiology. P wave is retrograde and negative in at least aVF.

4. QRS (RR) intervals

- a. Sinus rhythm/sinus node reentry: QRS (RR) intervals are regular.
- b. Sinus arrhythmia: QRS (RR) intervals are irregularly irregular.
- c. Atrial tachycardia, atrial flutter: QRS intervals are regular or regularly irregular, based on degree of AV block (and hence impulse conduction to the ventricle) that is present. The AV block may be fixed (*ie*, 2:1, 3:1, 4:1, etc.) or variable, including Wenckebach.
- d. Atrial fibrillation/MAT/WAP: The QRS (RR) intervals are irregularly irregular.
- e. Ectopic junctional tachycardia/ AVNRT/ AVRT: The QRS (RR) intervals are regular.

5. Response to drugs or enhanced vagal tone (carotid sinus pressure or Valsalva)

- a. Sinus tachycardia: There is a gradual decrease and then increase in rate after drug effect abates or after release of carotid sinus pressure or Valsalva.
- b. Sinus node reentry: There is no effect or abrupt termination.
- c. Atrial tachycardia (including multifocal atrial tachycardia), atrial flutter, atrial fibrillation: There is no change in atrial rate, but there may be the development of AV block and hence a slowing of ventricular rate.
- d. Ectopic junctional tachycardia, AVNRT, AVRT: There is no effect or an abrupt termination of arrhythmia.

6. Mode of termination

- a. Without atrial activity (termination with QRS only and no P wave or atrial activity following the last QRS complex of the tachycardia): atrial tachycardia, atrial flutter, atrial fibrillation, *ie*, rhythms generated within the atrial myocardium
- b. Nonconducted P wave (if P wave seen, termination with QRS complex and a P wave following the last QRS complex of the tachycardia): AVNRT, AVRT, and ectopic junctional tachycardia, *ie*, rhythms generated by or requiring the AV node.

Specific Supraventricular Tachyarrhythmias**1. Sinus tachycardia resulting from enhanced sympathetic tone or elevated levels of circulating catecholamines**

- a. A regular rhythm with P waves that are upright in leads I, II aVF, and V4–V6; the P wave is inverted in aVR.
- b. The rate is >100 bpm and there is only one P-wave morphology.
- c. Maximal rates are age-dependent (*ie*, 220 – age).

2. Sinus node reentry due to a reentrant circuit involving the sinus node and the tissue around the node (FIGURE 1)

- a. A regular rhythm with a P wave that is upright in leads I, II, aVF, and V4–V6.
- b. There is only one P-wave morphology.

- c. This tachycardia appears identical to sinus tachycardia but has an abrupt onset and an abrupt offset, in contrast to true sinus tachycardia, where the rate increases and slows gradually.
- 3. Atrial tachycardia most often is the result of a single ectopic focus in the atrial myocardium.**
 - a. The atrial rate is 100–220 bpm.
 - b. There are distinct P waves of uniform morphology before each QRS complex. If two sequential P waves are seen (as a result of AV block), there is an isoelectric (flat) baseline between P waves.
 - c. The P waves are usually different from those of sinus rhythm (inverted or biphasic in leads where the sinus P waves should be upright).
 - d. The PR interval is longer or shorter than that of sinus rhythm. AV block may be seen (*ie*, 2:1, 3:1, etc., or variable).
 - e. QRS intervals are regular or regularly irregular if variable AV block is present.
 - f. The PR interval may be constant or variable (with a pattern) if Wenckebach is present.
 - g. Variable PR intervals may also be seen as a result of concealed AV nodal conduction, *ie*, with the rapid atrial rate some atrial impulses get through the AV node, some are blocked, but some may partially penetrate the AV node and not traverse through completely (concealed within the node) yet may alter the rate of conduction of the subsequent atrial impulse, causing it to be conducted more slowly.
- 4. Multifocal atrial tachycardia resulting from multiple atrial ectopic foci; the atrial rate is >100 bpm; when the rate is <100 bpm it is termed wandering atrial pacemaker or multifocal atrial rhythm.**
 - a. There are P waves before each QRS complex. However, there are ≥3 different P-wave morphologies and no P-wave morphology is dominant.
 - b. PR intervals vary.
 - c. PP and QRS (RR) intervals are irregularly irregular.
- 5. Typical atrial flutter results from a reentrant circuit in the right atrium that is the result of an anatomic block (*ie*, isthmus dependent) (FIGURE 1); the atrial rate is 260–320 bpm.**
 - a. Atrial flutter waves are negative/positive in leads II, III and aVF. The flutter waves are completely uniform in morphology, amplitude and interval.
 - b. There is no isoelectric baseline between flutter waves; they are continuously undulating (sawtooth) reflecting continuous electrical activity resulting from impulse conduction around the reentrant circuit or impulse conduction between the right and left atria.
 - c. QRS intervals are regular.
 - d. The atrial flutter rate may be slower as a result of antiarrhythmic drugs or disease of the atrial myocardium; however, the waveforms maintain a typical flutter morphology.

- e. The RR intervals are regular; however, if variable AV block is present (including Wenckebach) the RR intervals may be irregular with a pattern reflecting AV block (*ie*, the rhythm is regularly irregular). If the AV block is variable, including Wenckebach, the QRS intervals will be regularly irregular, based on the degree of AV block.
 - f. There may be a variable relationship between flutter wave and QRS complex due to concealed AV nodal conduction; *ie*, with the rapid atrial rate some atrial impulses get through the AV node, some are blocked, but some may partially penetrate the AV node and not traverse through completely (concealed within the node) but alter the rate of conduction of the subsequent impulse, causing it to conduct more slowly.
- 6. Atypical atrial flutter results from a reentrant circuit in the right atrium that is the result of a functional change in myocardial electrophysiologic properties and not an anatomic block (FIGURE 1); the atrial rate is >320 bpm.**
- a. The flutter waves are often positive in leads II, III, and aVF and they are completely uniform in morphology, amplitude, and interval.
 - b. There is no isoelectric baseline between flutter waves; they are continuously undulating (sawtooth) as a result of continuous electrical activity resulting from impulse conduction around the reentrant circuit or impulse conduction between the right and left atria.
 - c. QRS intervals are regular; however, variable AV block may be present (*ie*, 2:1, 3:1, 4:1, etc or Wenckebach). The QRS intervals will be regularly irregular if variable AV block is present.
- 7. Atrial fibrillation results from multiple small reentrant circuits in the atrial myocardium; the atrial rate is >320–450 or even more rapid.**
- a. There is no organized atrial activity; hence there is no distinct P wave. Rather, there are fibrillatory waves that are irregular in morphology, amplitude, and interval.
 - b. The fibrillatory waves may be coarse (>2 mm in amplitude) when atrial fibrillation is of recent onset or fine when atrial fibrillation is of longer duration.
 - c. The QRS intervals are irregularly irregular and the heart rate depends upon AV nodal conduction.
 - i. The normal AV node is generally capable of conducting each impulse up to rates of 170 bpm.
 - ii. Ventricular rates >200 bpm suggest an increased sympathetic state, while rates <100 bpm suggest AV nodal disease, the use of an AV nodal blocking drug, or enhanced vagal tone.
- 8. Junctional tachycardia is from an ectopic focus in the AV node or junction.**
- a. The rate is 100–220 bpm.
 - b. There is no P wave in front of any QRS complex.
 - i. An inverted (retrograde) P wave may be present following the QRS complex (due to VA conduction) with a stable R-P interval, which may be short (short RP tachycardia) or long (long RP tachycardia).

- ii. The P wave will always be negative in lead aVF, which is the lead that is perpendicular to the atria.
 - c. The QRS intervals are regular and QRS morphology is similar to that of sinus rhythm, although junctional tachycardia may be associated with rate-related aberration.
9. Typical atrioventricular nodal reentrant tachycardia (AVNRT) is a regular supraventricular tachycardia at a rate of 140–220 bpm.
- a. AVNRT is the result of dual AV nodal pathways within the AV node (FIGURE 1 and FIGURE 3).
 - b. There is a fast pathway that conducts rapidly but has a long refractory period (*ie*, long time to repolarize and recover) and a slow pathway that conducts slowly but has a short refractory period and short time to repolarize and recover.
 - c. These two pathways are linked proximally in the atrial myocardium and distally in the bundle of His, forming a circuit within the AV node.
 - d. A typical AVNRT is initiated by a premature atrial impulse that arrives at the AV node before the fast pathway has recovered and hence is conducted to the ventricle via the slow pathway. The premature complex has a long PR interval. Since impulse conduction is slow, the impulse may arrive at the distal portion of the circuit when the fast pathway has recovered or repolarized and, therefore, will conduct retrogradely via the fast pathway back to the atrium.
 - i. If the slow pathway has recovered or repolarized when the impulse reaches the proximal part of the

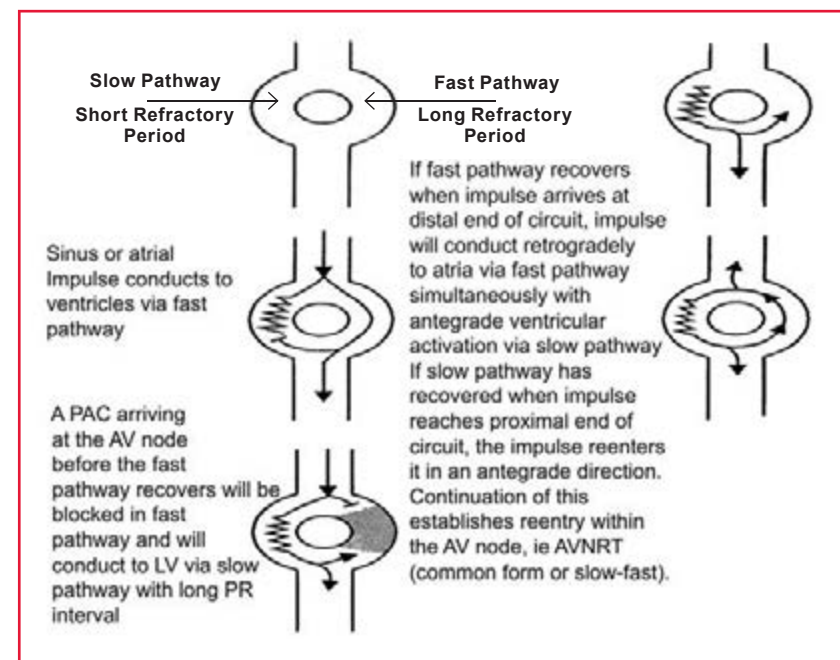


Figure 3. The electrophysiologic mechanism for the occurrence of atrioventricular nodal reentrant tachycardia. The anatomic etiology is the presence of dual AV nodal pathways, *ie*, a fast pathway that has a long refractory period and a slow pathway that has a short refractory period.

circuit, this impulse may reenter the slow pathway. If this process continues, a reentrant arrhythmia, *ie*, AVNRT, is established. In this case it is termed slow-fast (*ie*, the impulse conducts antegradely via the slow pathway to activate the ventricles at the same time that retrograde conduction via the fast pathway results in atrial activation).

- ii. Hence there is simultaneous or almost simultaneous ventricular and atrial activation, and there is usually no retrograde P wave seen after the QRS complex or the P wave may be superimposed on the end of the QRS complex and not distinguishable as a P wave, but appearing to be a R' or S wave.
- iii. A variation of a typical AVNRT is a slow-slow form, in which the antegrade activation of the ventricle is via a slow pathway while the retrograde conduction to the atrium uses the fast pathway, which conducts somewhat slowly (as a result of some AV nodal disease process or a drug). As a result, there will be a P wave seen shortly after the QRS complex with a short RP interval.

10. Atypical AVNRT, which is termed fast-slow, has a retrograde P wave seen after the QRS complex with a long RP interval.

- a. This is due to rapid antegrade conduction to the ventricles via the fast pathway while there is slow retrograde conduction to the atria using the slow pathway.
- b. An atypical AVNRT is often induced by a premature ventricular complex in which the retrograde impulse blocks in the fast pathway, but conducts retrogradely to the atria via the slow pathway. If it reaches the proximal portion of the circuit when the fast pathway has recovered, it will conduct antegradely to the ventricles via the fast pathway. If it reaches the terminal portion of the circuit when the slow pathway has recovered, it may reenter the slow pathway. If this continues atypical AVNRT is established.

- 11. Atrioventricular reentrant tachycardia (AVRT) is a regular supraventricular tachycardia associated with overt bypass tract as seen in a preexcitation syndrome, either Lown-Ganong-Levine (short PR interval and normal QRS complex) or Wolff-Parkinson-White (short PR interval and wide QRS complex with delta wave) (FIGURE 1). It also occurs in association with a concealed bypass tract (normal PR interval and normal QRS complex). Concealed bypass tract usually only conducts in a retrograde direction.**

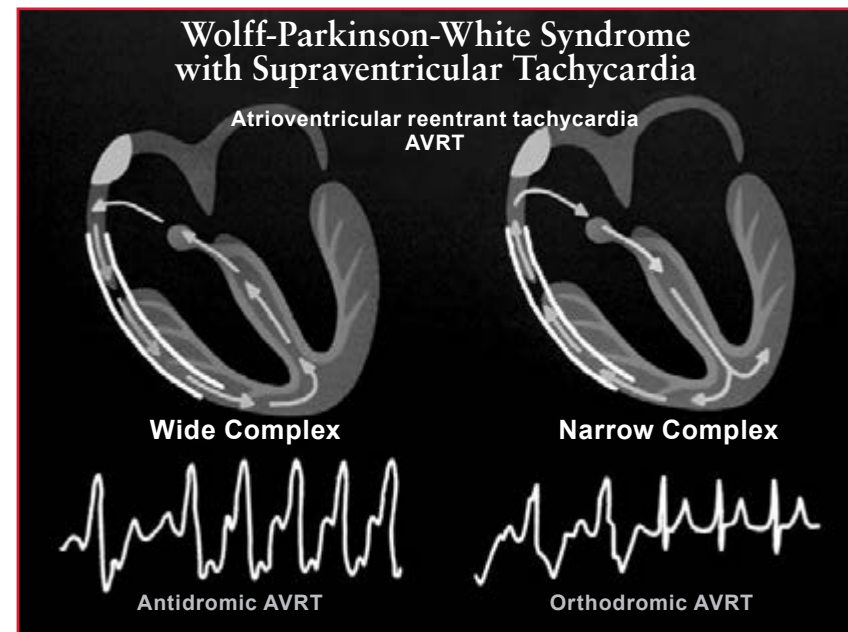


Figure 4. Anatomic pathways in Wolff-Parkinson-White involved in the development of orthodromic or antidromic atrioventricular reentrant tachycardia. Both the accessory pathway and the normal AV node–His–Purkinje pathway conduct an impulse from the atria to the ventricles. These two pathways are linked proximally in the atria and distally in the ventricular myocardium forming a macroreentrant circuit.

- a. The accessory pathway is a second pathway (along with the AV node–His-Purkinje pathway) linking the atrium and ventricles. As a result there is a macroreentrant circuit.
- b. In Wolff-Parkinson White, the accessory pathway (bundle of Kent) goes from the atrial myocardium directly with the ventricular myocardium (FIGURE 1).
- c. In Lown-Ganong-Levine the accessory pathway (bundle of James) goes from the atrial myocardium into the bundle of His (FIGURE 1).
- d. The usual rate of AVRT is 140–240 bpm.
- e. Most commonly, it presents as a short PR tachycardia, *ie*, there is a retrograde P wave (negative P wave in at least aVF) after each QRS complex with a short RP interval that is constant.
 - i. It is less common to have a long RP tachycardia.
 - ii. Rarely it may present as a no-RP tachycardia.
- f. It is less common to have a long RP tachycardia and rarely it may present as a no-RP tachycardia.
- g. Wolff-Parkinson-White there are two forms of an AVRT (FIGURE 4):
 - i. Orthodromic AVRT: In this situation, there is antegrade conduction to the ventricle via the normal AV node–His-Purkinje pathway. The retrograde conduction back to the atrium is via the accessory pathway. As ventricular activation is via the normal conduction system, the QRS complex is narrow and normal in morphology. However, there may also be an associated rate-related bundle branch block, which results in a wide QRS complex that has a typical right or left bundle branch morphology. The QRS morphology will not resemble that of the preexcited sinus complex.
 - ii. Antidromic AVRT: In this situation, the antegrade conduction to the ventricle uses the accessory pathway, while there is retrograde conduction back to the atrium via the normal His-Purkinje-AV nodal pathway. Since there is direct ventricular myocardial activation via the accessory pathway, and not the normal His-Purkinje system, the QRS complex is wide and abnormal. It might be difficult to distinguish between antidromic AVRT and ventricular tachycardia, which is also due to direct ventricular myocardial activation and an abnormal QRS complex morphology. The most important feature of antidromic AVRT is that the QRS complex of the tachycardia will have a morphology that is identical to that of the aberrated or preexcited QRS during sinus rhythm (WPW pattern). It may be wider, however, because it will be maximally preexcited.
- h. On occasion there may be a concealed bypass tract. However, these pathways only conduct retrogradely, from left ventricle to atrium. Antegrade conduction to the ventricle will be via the AV node–His-Purkinje system, while retrograde conduction to the atrium is via the bypass tract. Hence the AVRT will be orthodromic and will have a narrow or normal QRS complex morphology.

- i. In Lown-Ganong-Levine, the AVRT has a QRS complex that is narrow and normal in morphology regardless of whether antegrade conduction to the ventricle is via normal His-Purkinje pathway or via the accessory pathway. This is because the accessory pathway goes from the atrium and links into the bundle of His. Therefore, regardless of which pathway conducts antegradely to the ventricle, the normal His-Purkinje system is used to activate the ventricles. Depending upon which pathway is used for ventricular activation, retrograde activation of the atria is via either the AV node or the accessory pathway.

Wide QRS Complex Tachyarrhythmia

A wide complex tachyarrhythmia is defined as a tachycardia (heart rate >100 bpm) with a wide QRS complex (≥ 120 msec), which may be due to a right bundle branch block, left bundle branch block, nonspecific intraventricular conduction delay, or direct ventricular activation that bypasses the normal His-Purkinje system, *ie*, a ventricular complex or a preexcited complex.

Etiologies of a Wide Complex Tachycardia

1. Ventricular tachycardia
2. Any supraventricular rhythm (sinus tachycardia, atrial tachycardia, atrial flutter, atrial fibrillation, AVNRT) with rate-related aberration or functional bundle branch block. Rate-related aberration may be due to:

- a. Underlying conduction system disease
- b. Slowing of conduction due to antiarrhythmic drug (class 1A or 1C)
- c. Hyperkalemia

3. Any supraventricular rhythm (sinus tachycardia, atrial tachycardia, atrial flutter, atrial fibrillation, AVNRT) with a preexisting intraventricular conduction delay or bundle branch block
4. Any supraventricular rhythm (sinus tachycardia, atrial tachycardia, atrial flutter, atrial fibrillation, antidromic AVRT) associated with an accessory pathway and preexcitation
5. Pacemaker-associated tachycardia
 - a. Pacemaker-mediated tachycardia (endless loop tachycardia). This occurs when there is a dual chamber pacemaker and intact VA conduction resulting in retrograde atrial activation. If the atrial lead senses this atrial impulse, it will pace the ventricle; if VA conduction continues, then a reentrant form of arrhythmia is established (*ie*, endless loop tachycardia).
 - b. Pacemaker tracking of atrial arrhythmia, *ie*, A sensed V paced

ECG Clues to Differentiate the Mechanism of a Wide QRS Complex Tachyarrhythmia

Importantly, supraventricular tachyarrhythmias, regardless of etiology, generally have 1:1 atrial-ventricular relationship (and hence AV dissociation is not seen) and the conduction of each electrical impulse to the ventricle always uses the same pathway (AV node–His-Purkinje or an

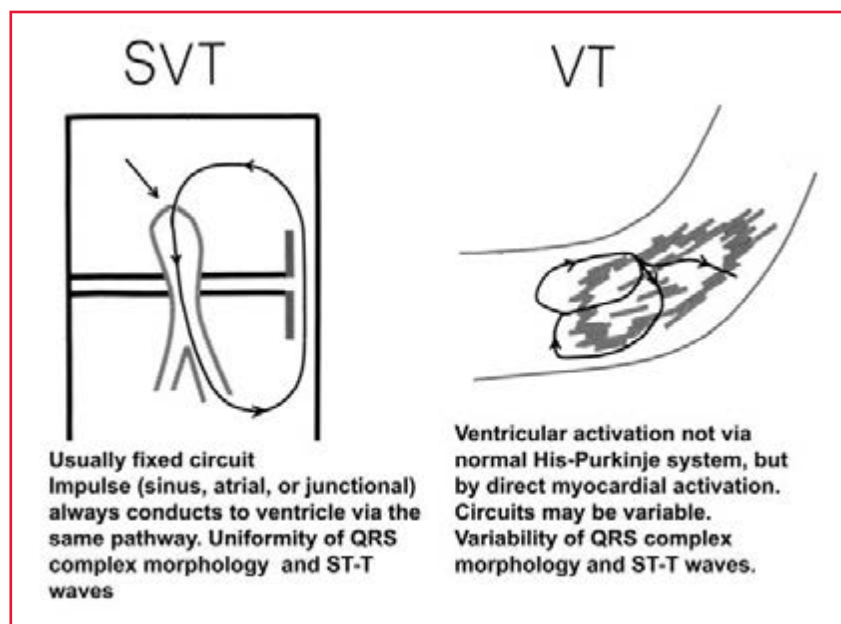


Figure 5. Pathways involved in supraventricular tachyarrhythmias and ventricular tachyarrhythmias. Each impulse associated with any supraventricular arrhythmia (sinus, atrial, or AV nodal) always conducts to the ventricle through the same pathway (*ie*, the normal AV node–His–Purkinje system or an accessory pathway). An impulse generated within the ventricular myocardium does not follow any fixed pathway and hence the direction of ventricular activation may be variable.

accessory tract) (FIGURE 5). Hence all the QRS complexes and ST-T waves have the same morphology. Ventricular tachycardia is due to a reentrant pathway within the ventricle, and ventricular activation bypasses the normal His–Purkinje system and is via an abnormal pathway and direct myocardial stimulation. Hence there is often AV dissociation (due to a rapid ventricular rate and inability to retrogradely penetrate the AV node to stimulate the atria) and also variability in the

QRS and ST-T wave morphologies. This is the result of slight changes in the direction of ventricular activation due to the variability of the ventricular activation sequence resulting from myocardial activation using a non-Purkinje pathway (FIGURE 5). This also results in changes in the direction of repolarization and changes in the ST-T waves. The ST-T wave changes may also reflect superimposed P waves.

The most important or useful ECG findings that are helpful for distinguishing ventricular tachycardia from a supraventricular tachycardia with aberration are:

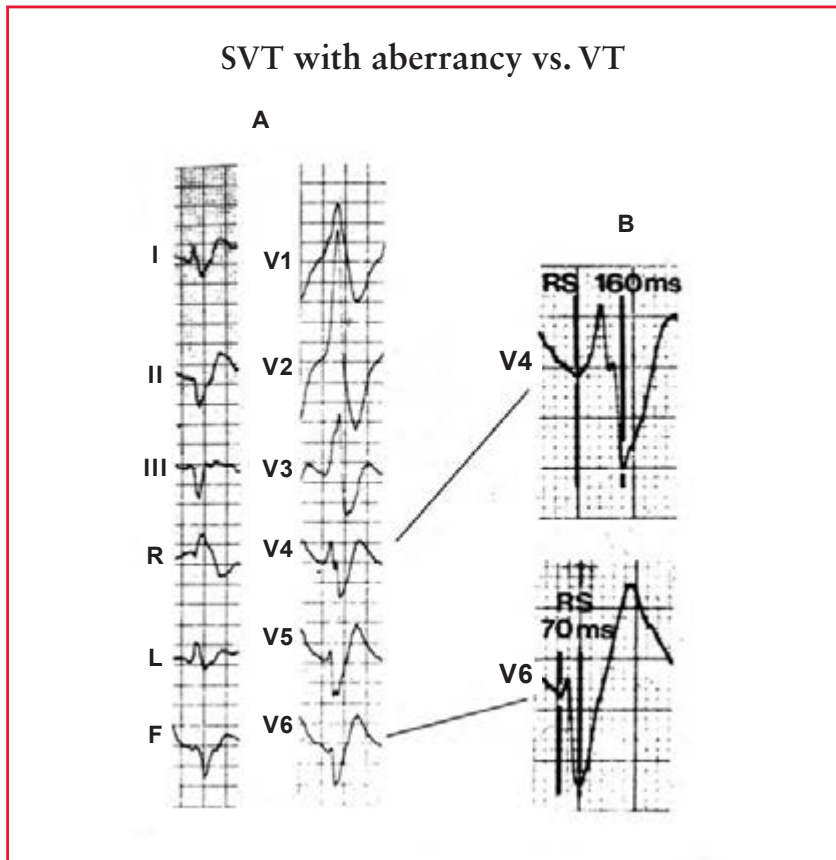
1. The presence of AV dissociation (*ie*, variable PR or RP intervals with no relationship between P waves or QRS complexes) and a ventricular rate faster than the atrial rate is the most important feature of ventricular tachycardia.
 - a. The P waves do not need to be “marched out.” Rather, the presence of a P wave associated with some, but not all of the QRS complexes, will establish AV dissociation.
 - b. It is rare for a supraventricular tachycardia to have AV dissociation.
 - c. Also supporting a diagnosis of AV dissociation is the presence of fusion or captured QRS complexes (also called Dressler beats). Fusion or captured QRS complexes are the result of intermittent antegrade conduction through the AV node, which results in partial (fusion) or complete ventricular capture, *ie*, there is fusion between an impulse conducted antegradely through the AV node and a complex generated from the ventricular myocardium.

- d. The presence of a fusion or completely captured QRS complex does not result in penetration of the reentrant circuit, and therefore the underlying ventricular tachycardia is not affected.
 - e. Fusion or captured complexes occur more often when the rate of the ventricular tachycardia is slower, resulting in less retrograde impulse conduction into the AV node from the ventricular complex, which allows for more antegrade conduction of an atrial impulse through the node.
- 2. Non-rate–related changes or variability of QRS morphology, which is often subtle, may be seen.**
- a. In a supraventricular tachycardia (regardless of the etiology, *ie*, sinus, atrial, or AV nodal), the activation of the ventricle is always via the same pathway, which may be the normal AV node–His–Purkinje system or an accessory pathway.
 - b. As the activation sequence is always the same, all the QRS complexes are identical to each other. In contrast, ventricular tachycardia is due to a small circuit within the ventricular myocardium and ventricular activation bypasses the normal Purkinje system.
 - c. As the vector of ventricular activation may change, there may be changes in the direction of ventricular activation or the myocardial activation sequence. Hence there may be subtle non-rate–related difference in QRS complex morphology.
 - d. Marked changes of QRS morphology and axis are diagnostic of polymorphic ventricular tachycardia.
- 3. Non-rate–related variability of ST segments and T waves that result from subtle differences in repolarization may be associated with subtle differences in depolarization sequence, or may be due to AV dissociation with superimposed P waves that occur in a variable fashion.**
- 4. Indeterminate axis (*ie*, QRS complex negative in leads I and aVF). This is not generally seen with any supraventricular complex as there is no form of aberration that is associated with an indeterminate axis. There are two reasons for an indeterminate axis:**
- a. The presence of two coexisting abnormalities—for example, a lateral wall myocardial infarction and a left anterior fascicular block, a lateral wall and an inferior wall myocardial infarction, a left posterior fascicular block and an inferior wall myocardial infarction, or right ventricular hypertrophy with a right axis and an inferior wall myocardial infarction or a left anterior fascicular block. In these cases the QRS complex has a supraventricular morphology.
 - b. With a wide QRS complex, an indeterminate axis may be seen in any situation in which there is direct ventricular activation that bypasses the normal His–Purkinje system. This includes a ventricular complex, a paced QRS complex (most often with biventricular pacing) or Wolff–Parkinson–White.

5. Also of importance is a major shift in axis compared to the sinus QRS complex. A significant shift in the QRS complex axis, particularly a significant shift to the left axis, is suggestive, but not diagnostic, of ventricular tachycardia. A shift of the axis rightward or a normal axis does not favor one diagnosis over another.
6. Positive concordance, *ie*, tall R waves across the precordium (V1–V6)
 - a. There is no form of aberration that will be associated with positive QRS complex concordance. This is seen in any other situation in which there is direct ventricular activation that bypasses the normal His-Purkinje system, including a ventricular complex, a paced QRS complex, or Wolff-Parkinson-White.
 - b. In contrast, negative QRS concordance (*ie*, deep QS complexes in leads V1–V6) can be seen with a typical left bundle branch block pattern. Hence negative concordance is not as useful.
7. A QRS complex width >160 msec is not usually seen with a bundle branch block, but it may be seen with a ventricular complex.
 - a. Exceptions are the presence of a dilated cardiomyopathy, in which the QRS complex interval may be as long as 200 to 220 msec, or the presence of hyperkalemia, which may cause widening of a supraventricular QRS complex to >160 msec.
 - b. A QRS complex that is wider than 240 msec is only seen with hyperkalemia.
8. In general, aberration is due to a terminal delay in ventricular activation (*ie*, either a right or left bundle branch block) that results in delayed activation of the ventricle served by that bundle. The initial forces of the QRS complex are, however, normal in width (*ie*, <0.10 sec) and morphology, as the initial ventricular activation still occurs via the normally conducting bundle and Purkinje system. The delayed activation to the right or left ventricles causes the terminal portion of the QRS complex (*ie*, R wave in V1 or S wave in V5–V6) to be prolonged.

In contrast, ventricular tachycardia is due to activation that does not use the normal Purkinje system, but rather there is direct myocardial stimulation. Direct myocardial stimulation results in slow conduction, and hence the entire QRS complex (including the initial portion), is wide, reflecting diffuse slowing of impulse conduction.

 - a. Thus, the presence of an R/S morphology in any precordial lead with an R wave width that is greater than the width of the S wave ($R/S > 1$) or an R wave >100 msec strongly suggests a ventricular complex or any other condition in which there is direct ventricular activation that bypasses the normal His-Purkinje system, such as a paced QRS complex or in Wolff-Parkinson-White (FIGURE 6). An $R/S > 1$ may also be seen with a dilated cardiomyopathy in which there is diffuse slowing of conduction through the ventricular myocardium.



Brugada et al *Circ* 83:1649, 1991.

Figure 6. Relationship between R and S wave width that helps in establishing the etiology of a wide QRS complex as being supraventricular with aberration or ventricular.

9. Specific QRS morphology criteria in V1 and V6 are less useful as they may not be definitive, although they may suggest a specific etiology.

- Such relationships are generally based on a statistical correlation and hence there is a good deal of overlap.
- Importantly, morphologic criteria that might favor a ventricular complex may be seen when there is a significant intraventricular conduction delay present during sinus rhythm, limiting their usefulness.
- Moreover, they are less valid when trying to distinguish between a ventricular and a preexcited complex.

However, morphologic criteria that have been proposed include:

- A monophasic R or biphasic qR complex in lead V1 favors VT; this represents the lack of an RSR' pattern.
- A triphasic RSR' or RsR' complex (the so-called "rabbit-ear" sign) in lead V1 usually favors a supraventricular rhythm. As an exception, if the R wave (initial positive waveform) of the RsR' complex is taller than the R' (terminal positive deflection), then VT is suggested.
- An rS complex (R wave smaller than S wave) in lead V6 favors VT. In contrast, an Rs complex (R wave larger than S wave) in lead V6 favors a supraventricular rhythm.

- In contrast, an R/S ratio that is <1 or an R wave that is <100 msec means that the initial ventricular activation time is normal, and this strongly suggests aberration as the cause of the wide QRS complex (FIGURE 6).

- iv. A broad initial R wave of 40 msec duration or longer in lead V1 or V2 favors VT. In contrast, the absence of an initial R wave or a small initial R wave of less than 40 msec in lead V1 or V2 favors a supraventricular rhythm.
 - v. A slurred or notched downstroke of the S wave in lead V1 or V2, and a duration from the onset of the QRS complex to the nadir of the QS or S wave of ≥ 60 msec in lead V1 or V2 favors VT. In contrast, a swift, smooth downstroke of the S wave in lead V1 or V2 with a duration of < 60 msec favors a supraventricular complex.
 - vi. The presence of any significant Q wave or a QS complex in V6 is suggestive of VT. In contrast, the absence of a Q wave in V6 favors a supraventricular complex.
- c. The presence of hyperkalemia (which slows impulse conduction)
 - i. When there is an abnormality of impulse conduction, there is a rate-related widening of the QRS complex, known as a functional bundle branch block.
 - ii. Conduction through the His-Purkinje system is “all or none” (*ie*, the His-Purkinje system either conducts or it does not conduct).

Therefore, in the presence of a bundle branch block, the velocity of conduction will always be the same, regardless of the heart rate, as conduction through the His-Purkinje system may be slow, but consistently so at all heart rates. If one of the bundles is blocked, conduction through the other bundle is intact and will not change with heart rate. Therefore, the QRS complex widening is abrupt and the width remains constant.

- d. When there is slowing of conduction through the His-Purkinje system as a result of an intraventricular conduction delay or an antiarrhythmic drug, there may be progressive widening of the QRS complex as the rate increases.
 - i. In the presence of an antiarrhythmic drug, the progressive widening of the QRS complex with increasing heart rate is the result of the use-dependent effect of these agents (particularly class IC and IA drugs).

Aberration

Aberrancy, which is defined as a supraventricular QRS complex that has a width > 120 msec, may occur as a result of:

1. Diffuse slowing of conduction through the His-Purkinje system (intraventricular conduction delay) or block in either the right or left bundle branches (bundle branch block).

The abnormality of conduction (*ie*, slowing of conduction velocity) may be due to:

- a. Underlying conduction system disease
- b. An antiarrhythmic drug (that slows impulse conduction)

- ii. This is the result of a progressive increase in the blockade of the sodium channel at faster heart rates, resulting in a progressive reduction in the upstroke velocity of phase 0, which determines the velocity of impulse conduction.

2. Aberrancy may also occur as a result of prolongation in His-Purkinje system refractoriness.

- a. In general, His-Purkinje refractoriness is longer when the heart rate is slower and refractoriness shortens at faster heart rates, allowing for the His-Purkinje system to conduct at faster heart rates. This rate-related change in refractoriness is not the result of sympathetic stimulation or other factors, but is related solely to heart rate.
- b. When there is an abrupt change in heart rate, *ie*, going from a slow (long RR interval) to a fast heart rate (short RR interval), His-Purkinje refractoriness may not adjust abruptly, resulting in a longer refractoriness at a faster heart rate. As a result, the impulse may not be conducted through a part of the conduction system, resulting in a block and hence aberration.
 - i. This is known as the Ashman phenomenon, and it is identified by an aberrated complex following a long-short RR interval.
 - ii. If there is a long RR interval (slow heart rate), followed by a sudden increase in the ventricular rate, *ie*, short RR interval, the complex that ends the short interval may be widened or aberrated as a result of failure of the impulse to conduction through one of the bundles.
- iii. As the right bundle has a slightly longer refractoriness compared to the left bundle, the aberrated complex usually has a right bundle branch block morphology.
- iv. The Ashman phenomenon is most often seen during atrial fibrillation, as there is continuously changing RR intervals or heart rate. However, it may be seen whenever there is an abrupt increase in heart rate (shortening of RR interval), such as with the onset of an atrial arrhythmia such as atrial tachycardia, atrial flutter or atrioventricular nodal reentrant tachycardia.
- v. Although most often only one QRS complex demonstrates aberration, the Ashman phenomenon may persist for several QRS complexes. The continuation of the aberrancy is due to the fact that when there is a right bundle branch block, the impulse that is conducted via the left bundle branch may retrogradely penetrate the right bundle, causing the right bundle to maintain an increased refractoriness and hence produce a right bundle branch block that may persist for several complexes.

3. Aberration is also seen in a preexcitation syndrome, primarily Wolff-Parkinson-White pattern. In this situation there are two pathways that link the atria and ventricles (*ie*, the normal AV node–His–Purkinje system and an accessory pathway known as a bundle of Kent).
 - a. The impulse from the atrium is transmitted to the ventricles via both pathways and hence the resulting ventricular complex represents fusion between myocardial activation via these two pathways.
 - b. As the accessory pathway bypasses the AV node, conduction to the ventricles via this pathway is much quicker. As a result, there is a short PR interval (or actually PR segment) and a widened QRS complex.
 - c. The widened or aberrated QRS complex is due to the fact that initial ventricular activation occurs via the accessory pathway (and hence the term preexcitation).
 - i. As there is direct ventricular myocardial activation, the impulse is conducted via the myocardial cells and is therefore slower than conduction through the normal Purkinje system.
 - ii. As a result, the initial portion of the QRS complex is wide with a slow or slurred upstroke, known as a delta wave.
 - iii. The rest of ventricular depolarization is via the normal AV node–His–Purkinje system and the remainder of the QRS complex is narrow. This presents with a QRS complex that has a wide base and narrow apex.
 - iv. The width of the delta wave is determined by the conduction velocity via the AV node. If AV nodal conduction is slow, more of ventricular activation is via the accessory pathway, resulting in a shorter PR interval and a wider more prominent delta wave, *ie*, more preexcited. If AV nodal conduction is faster, less of myocardial activation is via the accessory pathway, resulting in a longer PR interval and a narrower and less prominent delta wave. Therefore, there may be variability of the QRS complex width and PR interval.
 - v. This may happen spontaneously, even in the absence of a change in heart rate as changes in autonomic tone may only affect AV nodal conduction. This is known as the “concertina effect.”
 - d. An aberrated or preexcited QRS complex in WPW may be seen with any supraventricular tachycardia, including sinus tachycardia, atrial tachycardia or atrial flutter. It may also be seen with an antidromic AVRT, as discussed above.
 - e. Aberration is also seen in WPW during atrial fibrillation. In this situation there is irregular atrial activity occurring at rates of up to 450–500 bpm. Therefore, the AV node and accessory pathway are being “bombarded” rapidly by impulses. Whichever pathway is used is random, depending upon the location of the waveform origin in relation to the location of the pathways and the refractoriness of the pathways.

f. Based on the AV nodal refractory period, the AV node usually cannot conduct at rates over about 170 bpm, although in the presence of sympathetic stimulation AV nodal conduction is enhanced and the ventricular response rate may be more rapid. The accessory pathway, however, often has a very short refractory period and is therefore capable of conducting atrial impulses at a much more rapid rate. If this should occur, the ventricle may be stimulated at rates well above 300–350 bpm. At these rates, even a ventricle with normal myocardium may develop ventricular fibrillation.

g. The important finding associated with atrial fibrillation in WPW is an absence of a relationship between rate and QRS complex width, *ie*, there may be narrow QRS complexes with short RR intervals and widened (aberrated or preexcited) QRS complexes with longer RR intervals.

h. In addition, there is non-rate-related variability in QRS complex width. This reflects the fact that the WPW complex is a fusion complex and the width may change depending upon the relative contribution of ventricular myocardial activation coming from the accessory pathway or the AV node–His–Purkinje system. ■

Core ECGs

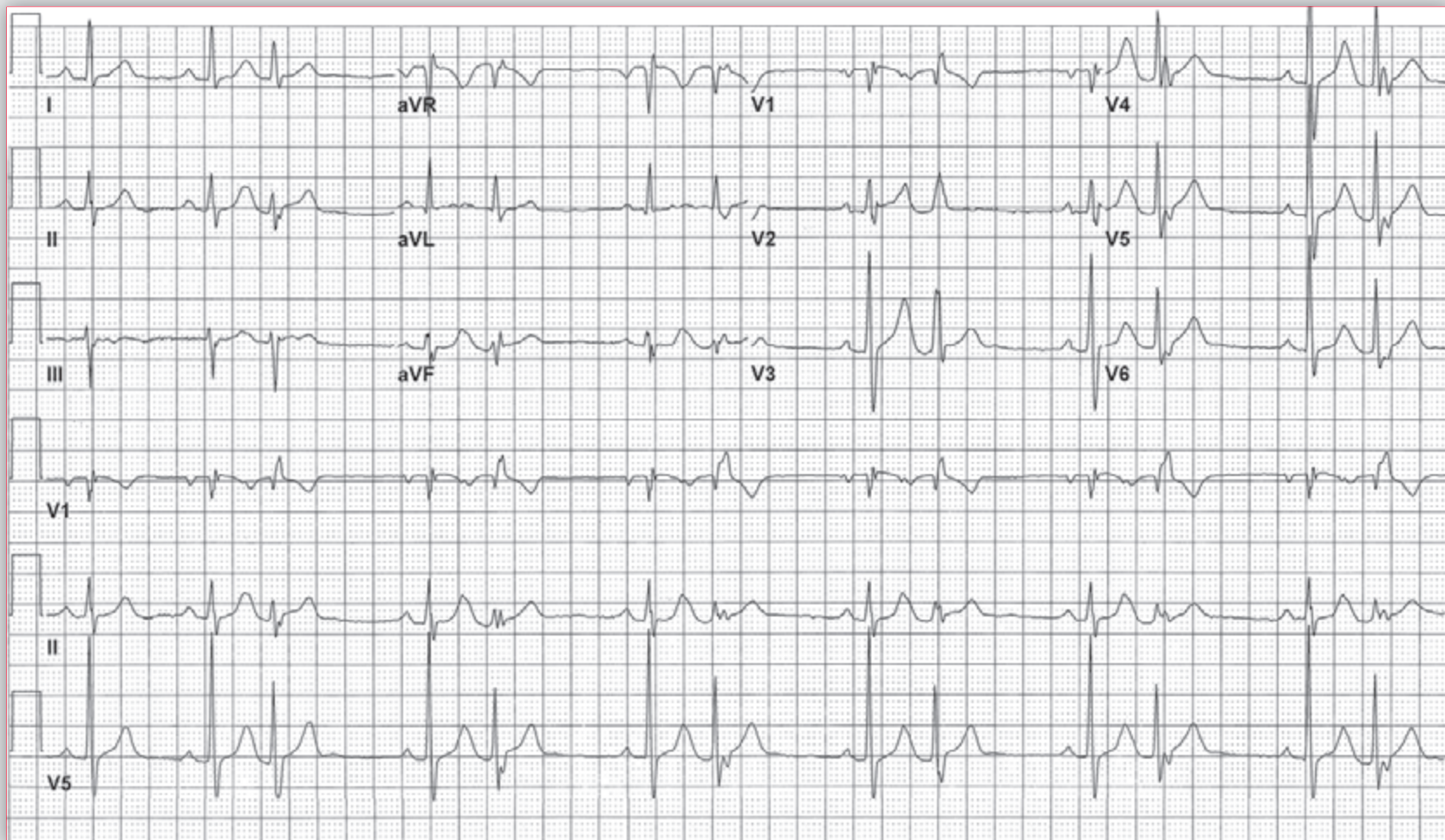
A 62-year-old male with prior history of hypertension presents to his primary doctor with a complaint of new-onset palpitations. The symptoms arise and abate without warning, and can last from a few minutes to an hour or so.

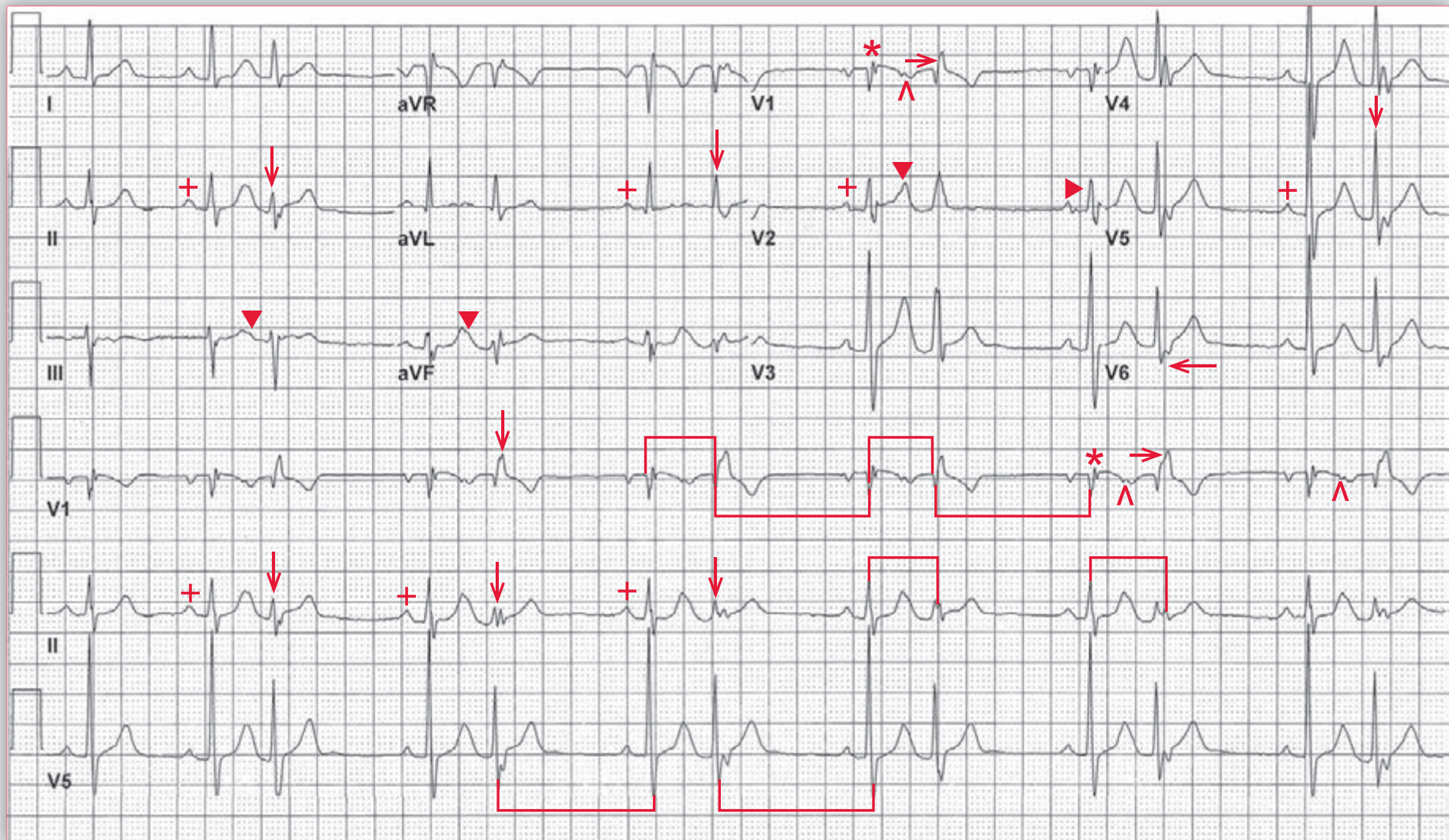
He notices them particularly in the late morning.

He denies any change in his functional status, specifically denying exertional chest pain and dyspnea. He denies presyncopal symptoms.

His physician obtains an ECG.

What abnormalities are noted that explain the patient's presentation?





ECG 1 Analysis: Normal sinus rhythm, early transition, premature atrial complexes in a bigeminal pattern (atrial bigeminy) with rate-related right bundle branch block

There is a regularly irregular rhythm with long (□) and short (□) RR intervals. In addition, there are narrow and wide QRS complexes. The overall rate is 78 bpm. A P wave can be seen before each narrow QRS complex (+), and there is a stable PR interval (0.16 sec). The P wave is upright in leads I, II, aVF, and V4–V6. Hence these are sinus complexes. The QRS complex duration is normal (0.10 sec) and there is a normal axis of about 0° (QRS complex positive in lead I and biphasic in lead aVF). The QT/QTc intervals are normal (390/450 msec). There is a small r' in lead V1 (*), representing a right ventricular conduction delay, and early transition with a tall R wave in V2 (►). This is due to counterclockwise rotation of the heart (*ie*, a change in the electrical axis in the horizontal plane). This is determined by imagining the heart as being observed from under the diaphragm. When there is counterclockwise electrical rotation, left ventricular forces develop earlier in the precordial leads (early transition), hence the tall R wave in lead V2.

After each sinus complex, there is an early or premature complex (↓) (accounting for the short RR interval) that is also preceded by a P wave (^), best seen in lead V1, but also seen notching the T waves in leads III, aVF, and V2 (▼). T waves should be smooth in upstroke and downstroke. Any notches, bumps, or other irregularities of the T waves

are very suggestive of superimposed P waves. The P-wave morphology is different from that of the sinus P wave. The PR interval is also longer (0.20 sec) than that of the sinus complex. Hence this is a premature atrial P wave. Following the premature P wave, there is a QRS complex that has a wider duration (0.12 sec) and has a morphology of a right bundle branch block, *ie*, RSR' in V1 (→) and broad S waves in leads I, V4–V6 (←). These are premature atrial complexes in a bigeminal pattern or atrial bigeminy (*ie*, every other QRS complex is a premature atrial complex); the QRS complex has a right bundle branch block aberration, representing a rate-related or functional bundle branch block.

The palpitations are probably the result of the frequent premature atrial complexes. The sensation of palpitations is generally the result of the pause following the premature complex, during which there is continued diastolic filling of the ventricles. With ventricular contraction, the increased end diastolic volume results in an increase in inotropy via the Starling effect. This increased myocardial contractility and increased stroke volume causes the sensation of palpitations. Premature atrial complexes are benign and do not require any specific therapy. The patient should be reassured that the palpitations are the result of a benign arrhythmia. ■

Notes

A 70-year-old man presents with complaints of occasional palpitations on most mornings. These episodes last a few hours and resolve spontaneously. He denies any associated symptoms. He is an active competitive golfer and has not noted a decrement in his ability to play 18 holes without a golf caddy or cart.

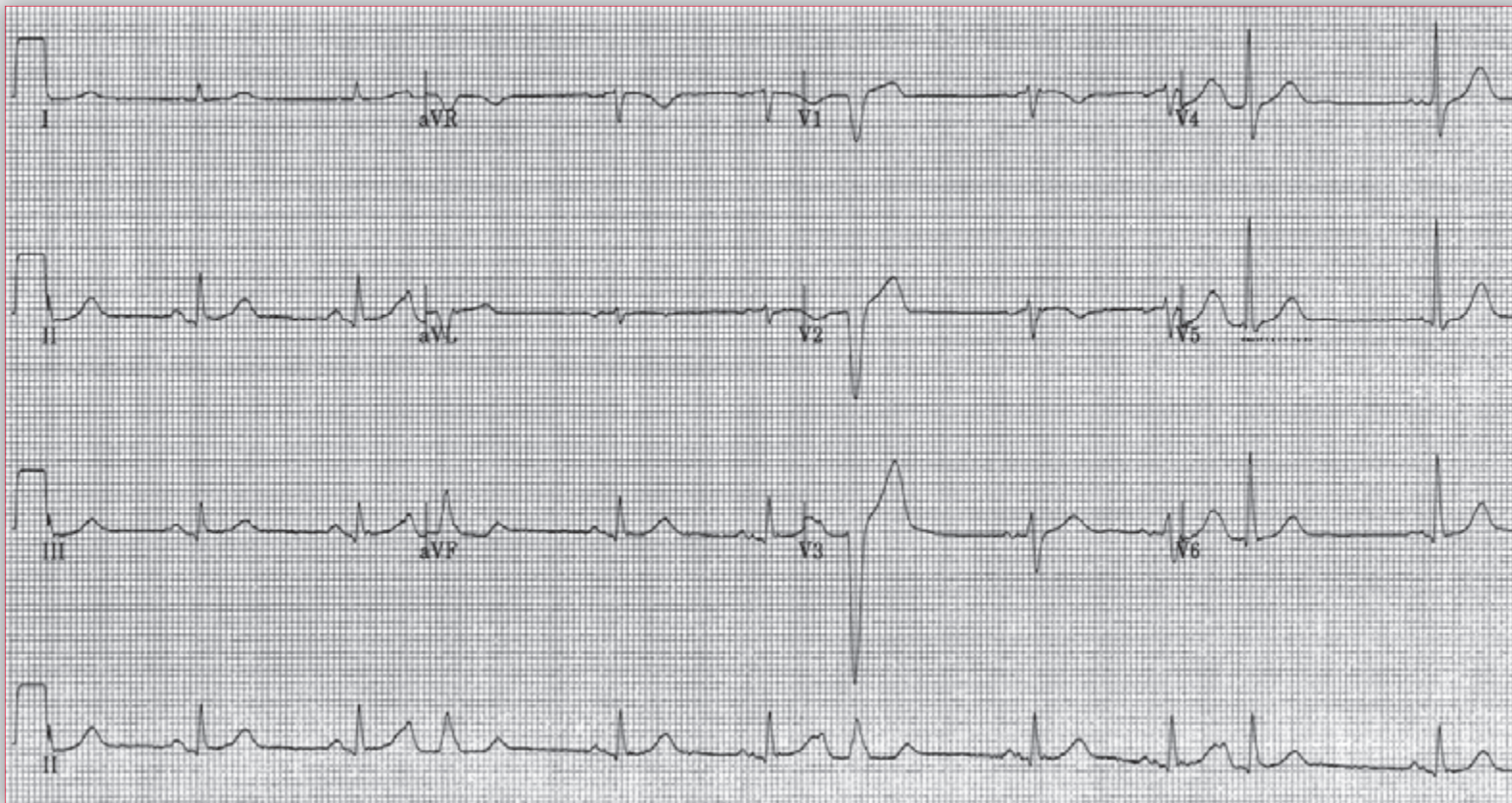
On further questioning, he does note that these symptoms only seem to occur on weekdays. He

notes that he is often at his office desk when the symptoms arise. For that reason, he thought the symptoms might be related to his morning coffee, which he drinks only on workdays. Prior to his visit with his physician, he consumed 3 cups of coffee in rapid succession.

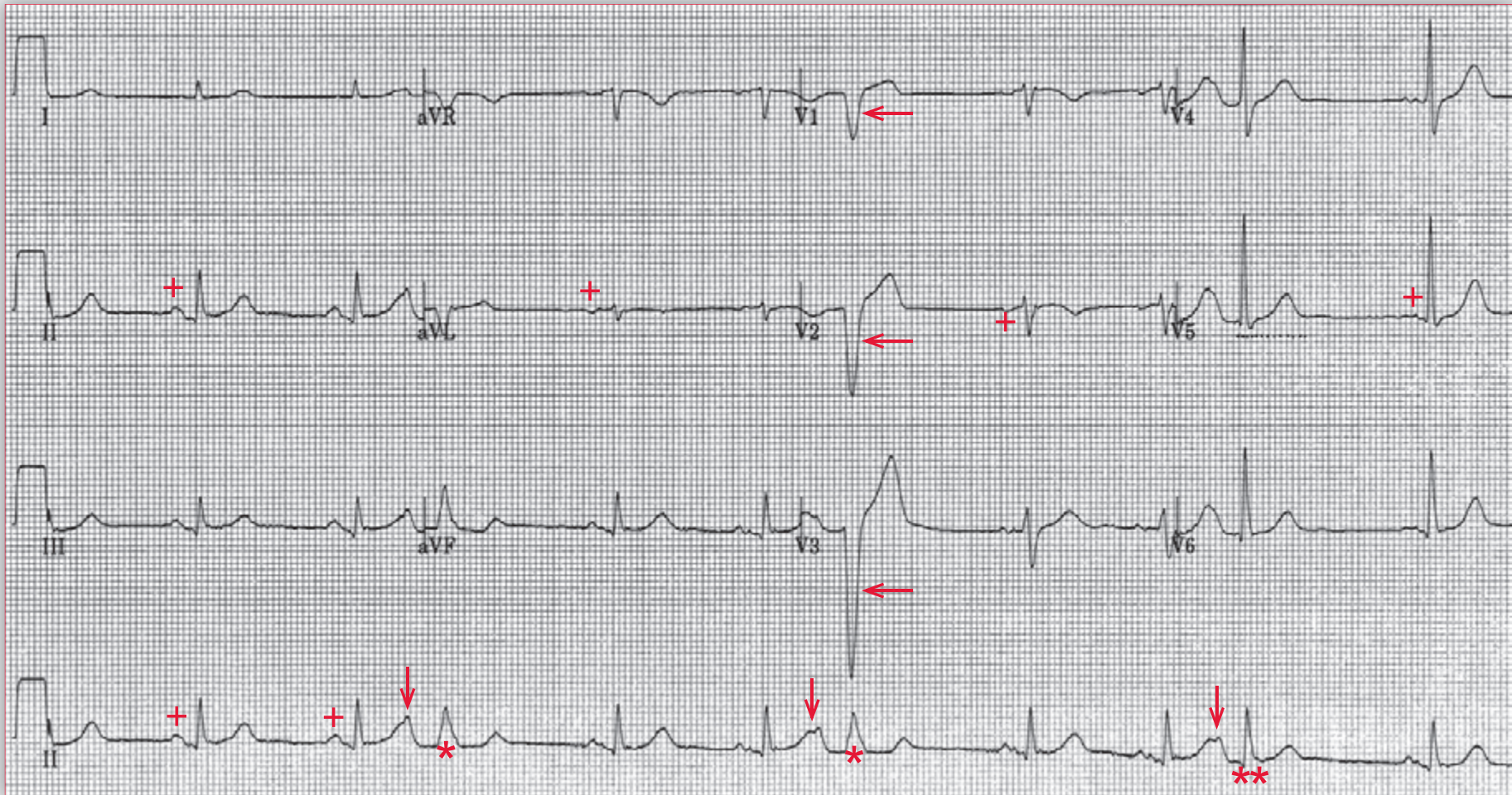
On exam, he is a fit-looking male, younger-looking than stated age. His physical exam is normal except for an irregular radial pulse.

On auscultation, the second heart sound loses physiologic splitting in time with the irregular beats. An ECG is obtained.

What is the cause of the patient's symptoms and how can the auscultatory findings be explained?



Podrid's Real-World ECGs



ECG 2 Analysis: Normal sinus rhythm, premature atrial complexes with rate-related left bundle branch block morphology

The rhythm is regularly irregular at a rate of 60 bpm. The irregularity is a result of three premature complexes (*). There is a P wave (+) before each QRS complex with a constant PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec) and there is a normal morphology and axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (400/400 msec). The third, sixth, and ninth QRS complexes are premature (*,**). A premature P wave (notch on the T wave) can be seen before each one (↓). T waves should have a smooth upstroke and downstroke and notches are usually the result of superimposed P waves. These are premature atrial complexes. Since every third complex is a premature atrial complex, this is termed atrial trigeminy.

The QRS complex duration of the third and sixth premature atrial complexes (*) is wide (0.16 sec) and there is a left bundle branch block morphology (broad R wave in II and aVF and a deep QR complex in

leads V1–V3 [←]). Hence these are premature atrial complexes with a left bundle branch block due to rate-related aberration or a functional bundle branch block. The last premature complex (**) (ninth complex) does not have a left bundle branch block aberration, likely due to the fact that the coupling interval between this premature complex and the previous sinus complex is slightly longer.

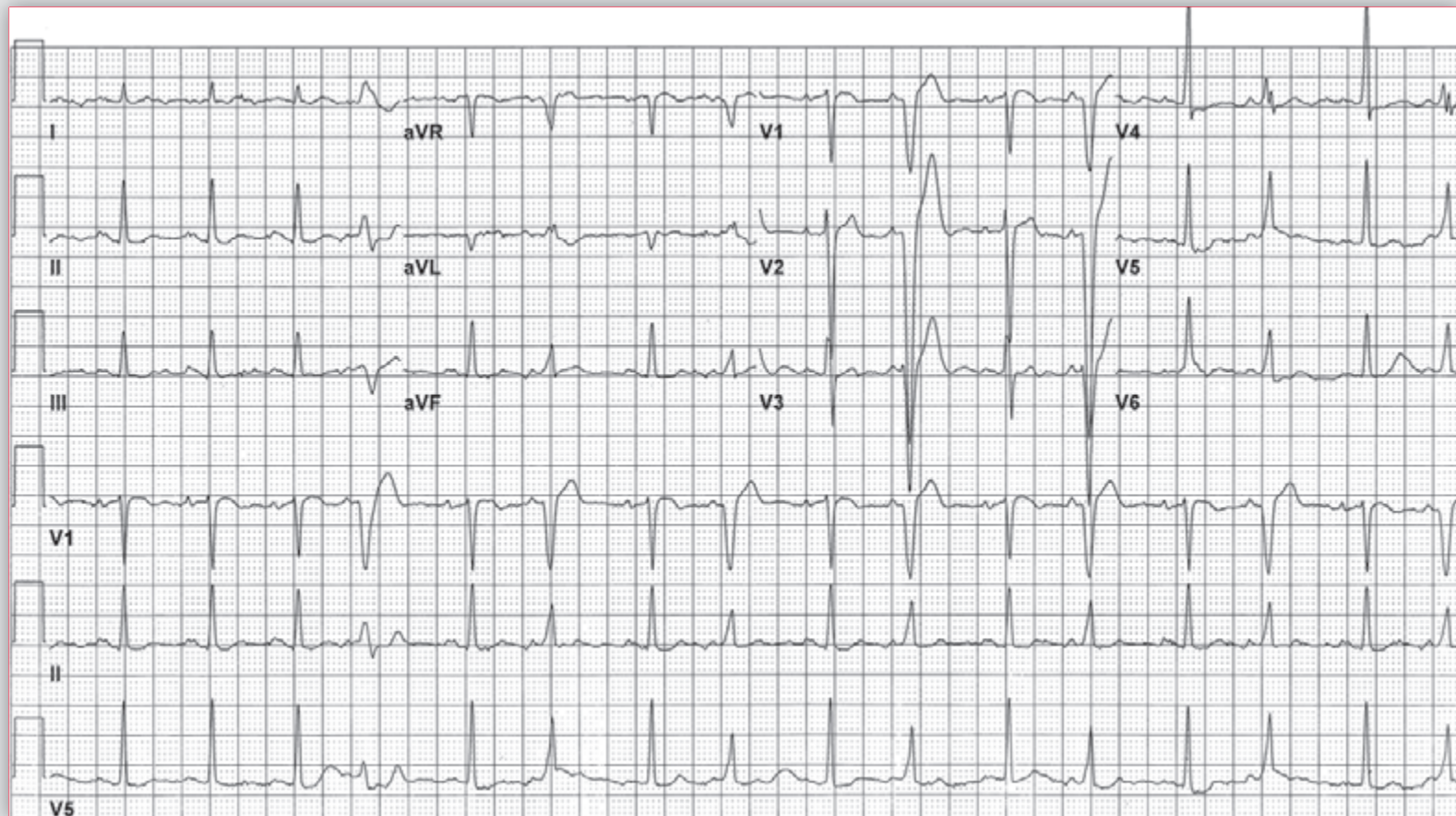
The normal two components of the second cardiac sound are that of the aortic valve closing followed by the pulmonic valve closing (A2→P2). During left bundle branch block, the closing of the aortic valve is delayed and A1 can move closer to, overlap, or even follow, P2 depending on the degree of conduction delay. Indeed, it is not uncommon for a left bundle branch block to be associated with paradoxical splitting of S2 since A2 follows P2. With inspiration, P2 is delayed and moves toward A2, resulting a reduction in the split. With expiration, P2 is earlier, moving away from A2 and hence producing a wider split. ■

Core Case 3

A 44-year-old man presents with an indolent onset of fatigue. He is an avid mountain biker but has noticed over the past few months that he is not able to complete the same bike routes he was able to 6 months ago. Despite trying to increase his training program, he has been unable to meet his own personal endurance goals.

He does not carry any medical diagnoses and does not take any medications. His family history is notable for a father who had a stroke at age 65.

ECG 3A



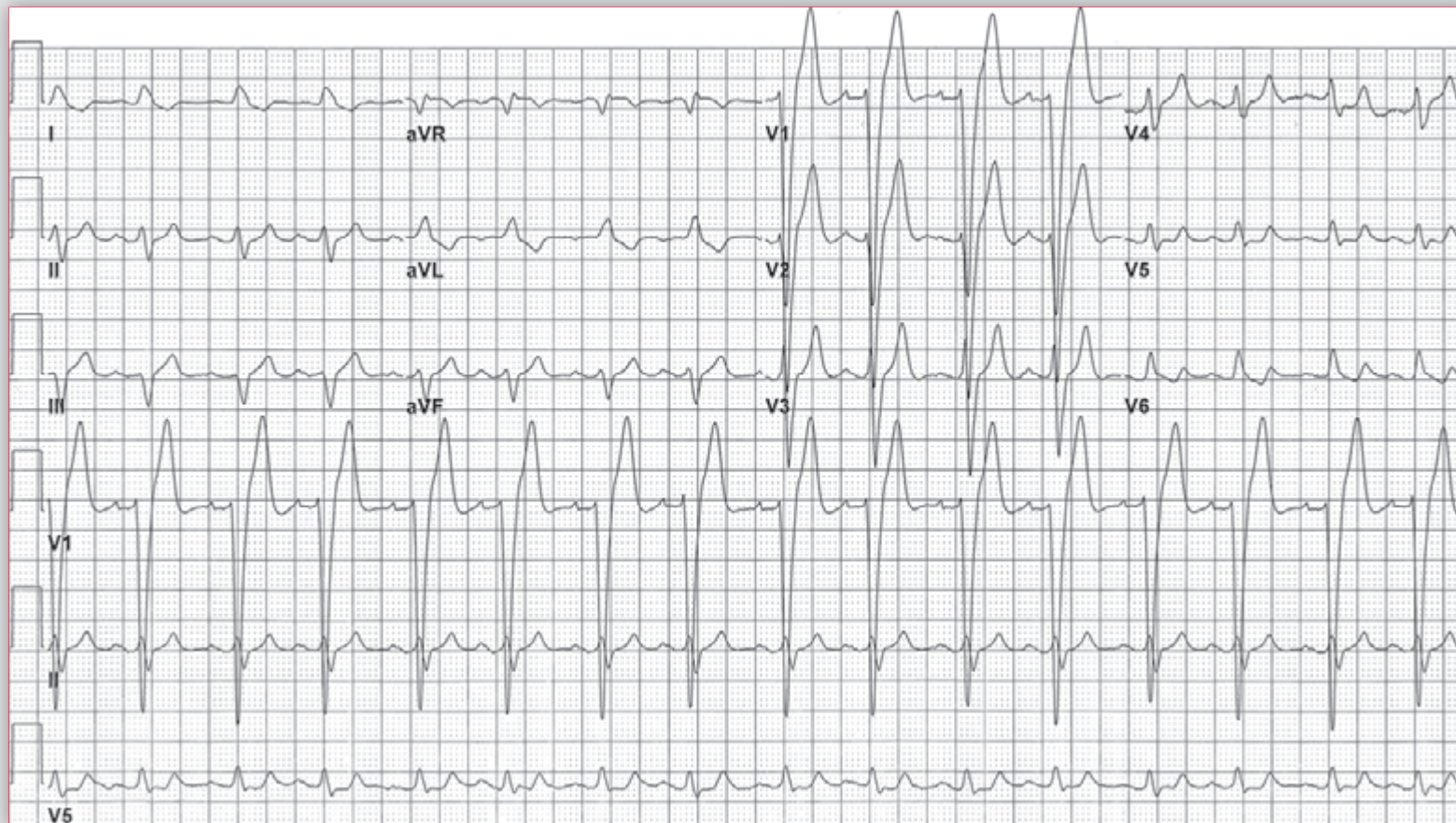
His review of systems is unremarkable.

As part of his workup, an ECG is obtained (ECG 3A).

He is seen in follow-up and a repeat ECG is obtained (ECG 3B).

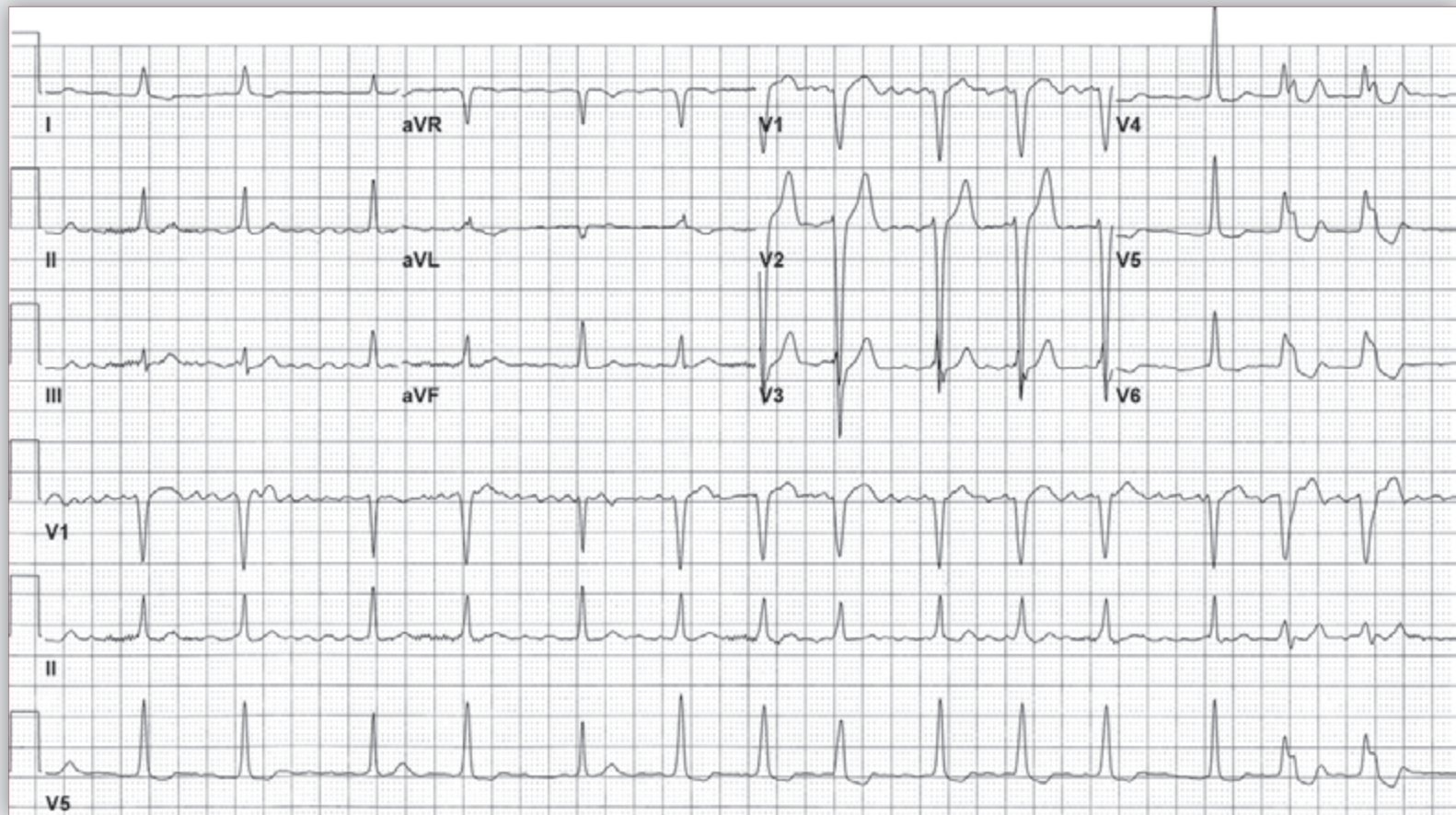
Several weeks later, he returns with marked progression of his fatigue and exertional dyspnea. The history of his present illness has not changed otherwise, and a thorough review is again unremarkable. An ECG is again obtained (ECG 3C).

ECG 3B



Core Case 3

ECG 3C

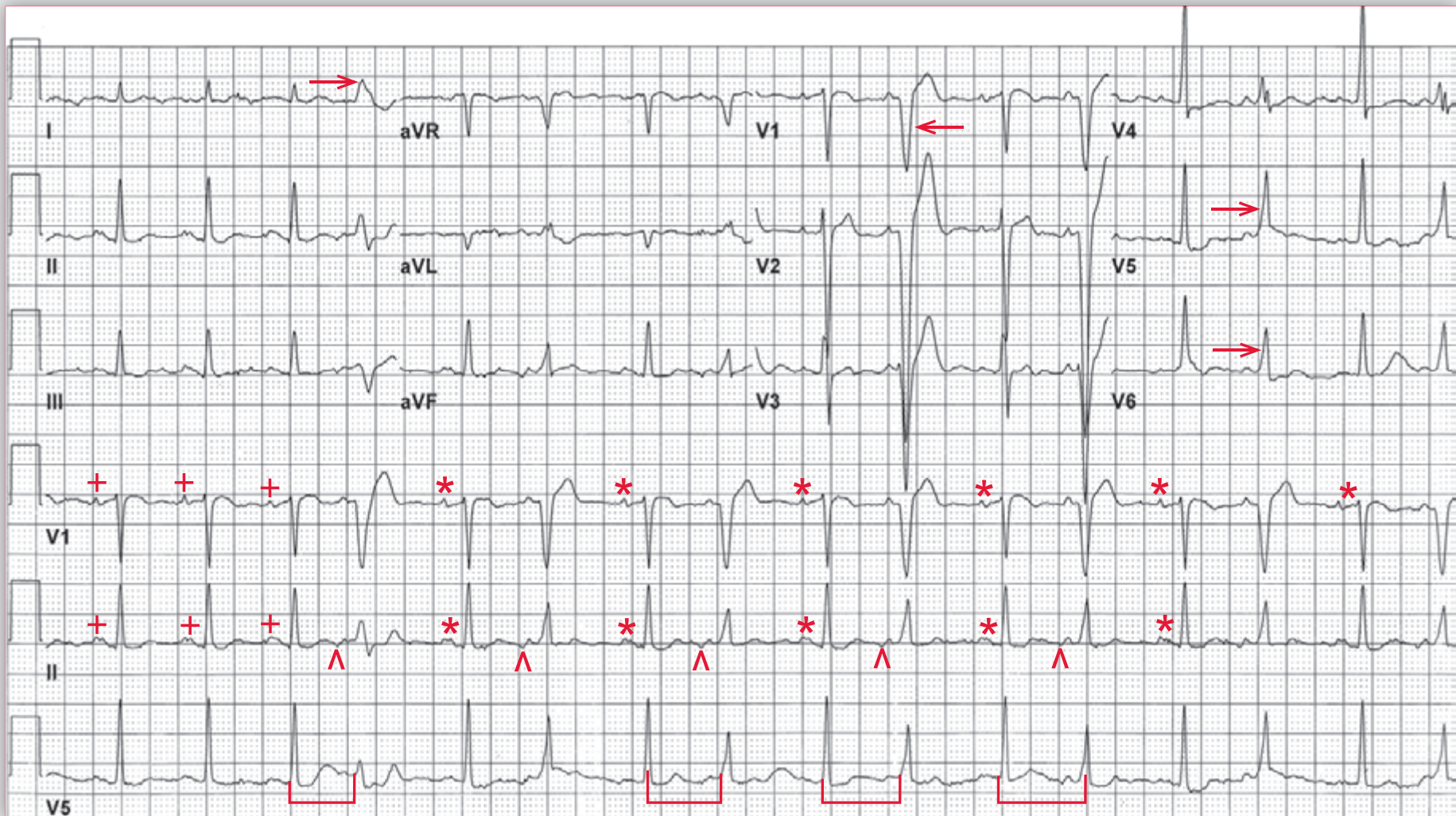


What abnormalities are noted on ECG 3A?

What further information does tracing (ECG 3B) provide?

What is the etiology of this patient's symptoms based on the details from ECG 3C?

Podrid's Real-World ECGs



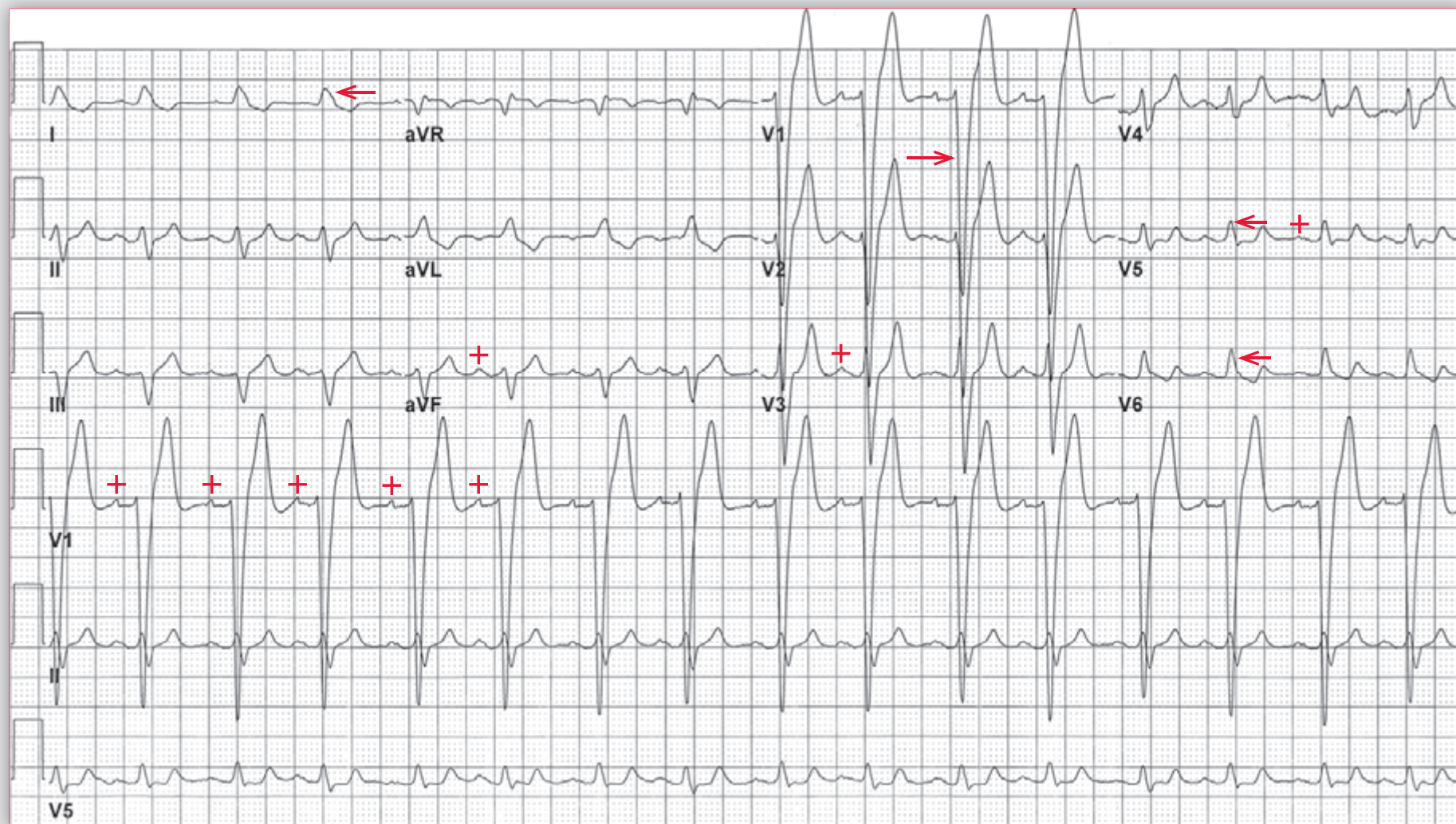
ECG 3A Analysis: Normal sinus rhythm, left atrial hypertrophy or abnormality (P-mitrale pattern), premature atrial complexes in a bigeminal pattern (atrial bigeminy) with a left bundle branch block (rate-related or functional bundle branch block)

ECG 3A shows a regularly irregular rhythm at a rate of 96 bpm. The first three QRS complexes have a normal duration (0.08 sec) at a regular rate of 100 bpm. There is a P wave (+) before each of these QRS complexes with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, and aVF. These are sinus complexes. The P wave is slightly broad and has a notching, especially seen in leads II and aVF; this is suggestive of left atrial hypertrophy or a left atrial abnormality. Thereafter every other QRS complex is narrow with a similar QRS morphology to the first three complexes. They are also preceded by a P wave (*) with a stable PR interval that is the same as the first three QRS complexes (0.16 sec). Hence these are sinus complexes. After each of these sinus complexes, there is a premature P wave (^) that has a different morphology (negative-positive in lead II) and a different PR interval (0.12 sec). This is a premature atrial P wave. Following the

premature P wave there is a premature wide QRS complex (0.14 sec) accounting for the short RR interval (⌐) and the irregularity. All the short RR intervals are the same, indicating a fixed coupling interval between the sinus complex and the premature atrial complex. The QRS complex has a pattern of a typical left bundle branch block (deep QS complex in lead V1 [←] and tall R wave in leads I and V4–V6 [→]). Hence these are premature atrial complexes in a bigeminal pattern (atrial bigeminy), and there is a rate-related left bundle branch block. There is a pause after each premature atrial complex, accounting for the long RR interval. With the slower rate, the sinus complex following the premature atrial complex has a normal duration (0.08 sec) and normal morphology and axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (300/380 msec).

continues

Podrid's Real-World ECGs



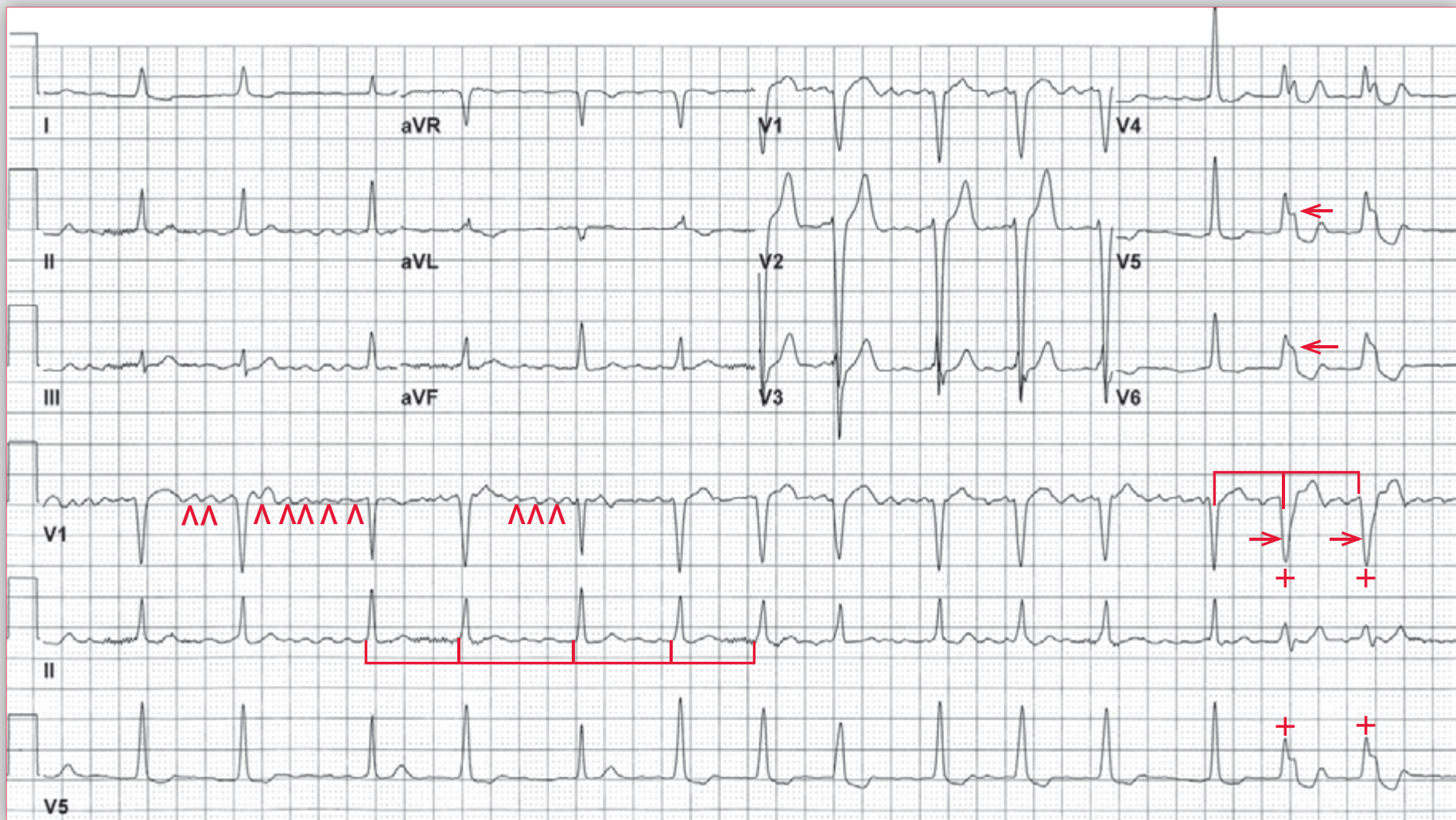
ECG 3B Analysis: Normal sinus rhythm, left bundle branch block

ECG 3B is from the same patient as ECG 3A. There is a regular rhythm at a rate of 98. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is prolonged (0.14 sec), and it has a left bundle branch block morphology (broad QS in lead V1 [→] and broad R wave in leads I and V5–V6 [←]) that is identical to the QRS complexes of the aberrated

premature atrial complexes in ECG 3A. As the rate is slightly faster than the sinus rate in ECG 3A, this is a rate-related left bundle branch block. The axis is about -30° (positive QRS complex in lead I, negative QRS complex in lead aVF and biphasic in lead II). The QT/QTc intervals are slightly prolonged 360/460 msec, but are normal when the prolonged QRS complex duration is considered (300/380 msec).

continues

Podrid's Real-World ECGs



ECG 3C Analysis: Atrial fibrillation, intermittent rate-related left bundle branch block

ECG 3C is for the same patient as ECGs 3A and 3B. However, the rhythm is now irregularly irregular and there are no obvious P waves seen before or after any of the QRS complexes. Hence this is atrial fibrillation, and the fibrillatory waves (which are irregular in morphology, amplitude and interval) can be seen in lead V1 and lead II (^). The QRS complexes duration is normal (0.08 sec) and the QRS axis and morphology are identical to the narrow QRS complexes in ECG 3A. The QT/QTc intervals are also the same. However, the last two QRS complexes (+) are wider and have a left bundle branch block morphology (deep QS in lead V1 [→] and broad R wave in leads V5–V6 [←]) that is the same as the QRS complexes in ECG 3B as well as the aberrated premature atrial complexes in ECG 3A. It can be noted that the RR intervals associated with the left bundle branch block morphology are shorter (□) than the RR intervals when the QRS complexes are narrow (□). Hence this is a rate-related left bundle branch block and

confirms that there is a rate-related left bundle branch block pattern seen with the premature atrial complexes in ECG 3A and the sinus rhythm in ECG 3B.

It is probable that the symptoms are the result of atrial fibrillation. With atrial fibrillation, there are a number of hemodynamic changes that can impact upon stroke volume and cardiac output and that can be associated with symptoms of fatigue and exertional dyspnea. These include: the loss of atrial contraction, which reduces left ventricular filling; a rapid ventricular rate, which reduces diastole and diastolic filling period; the irregularity of the RR intervals, which can alter and reduce the Starling effect; and a number of neurohormonal adaptations to the reduced stroke volume that can increase vascular resistance and further reduce stroke volume. This includes activation of the sympathetic nervous system and the renal-angiotensin-aldosterone system. ■

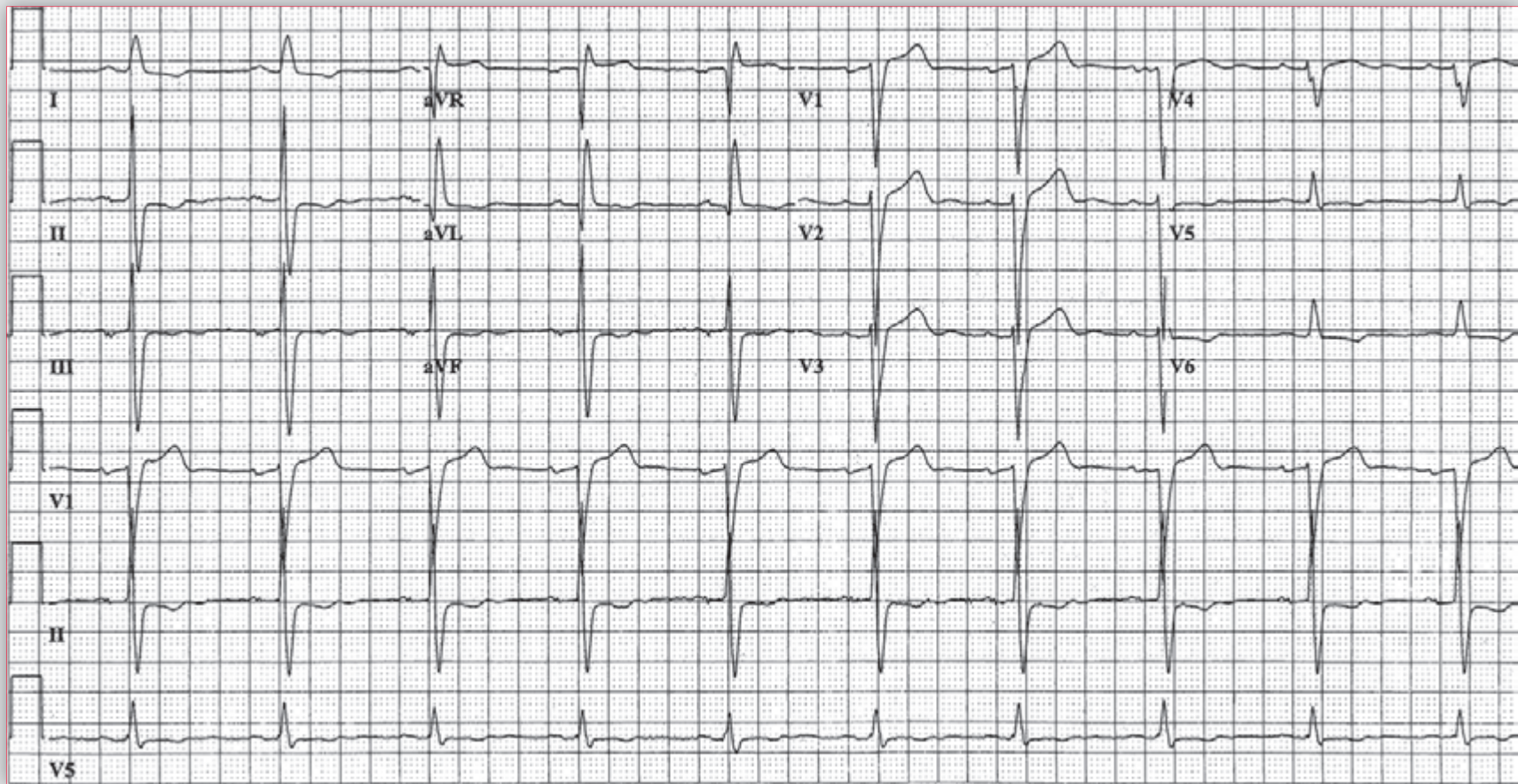
Core Case 4

A 44-year-old man with a history of alcohol abuse presents for an exercise treadmill test after he gives a history of new-onset exertional dyspnea and vague chest “heaviness.”

ECG 4A represents his baseline tracing.

ECG 4B represents his tracing during moderate exercise.

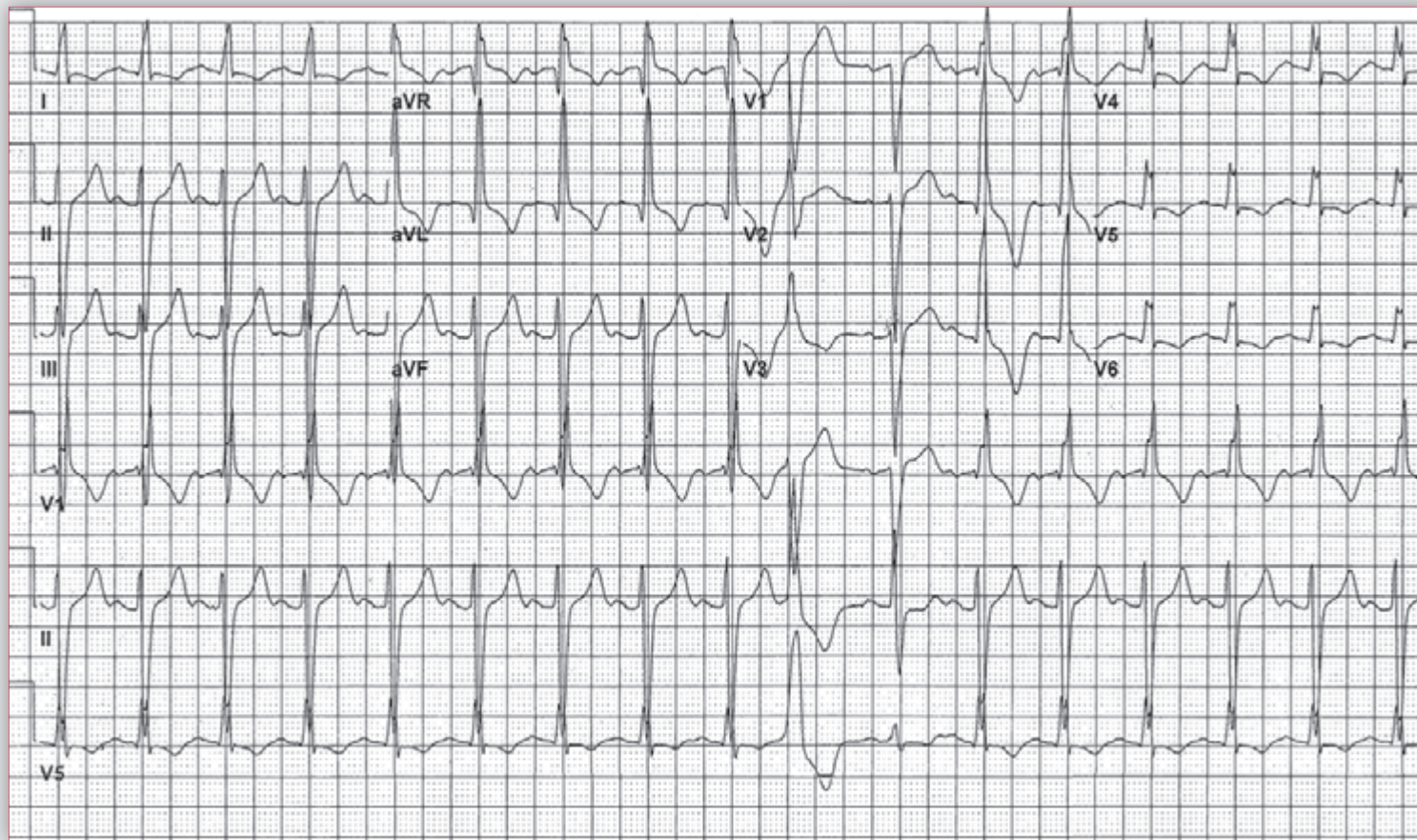
ECG 4A



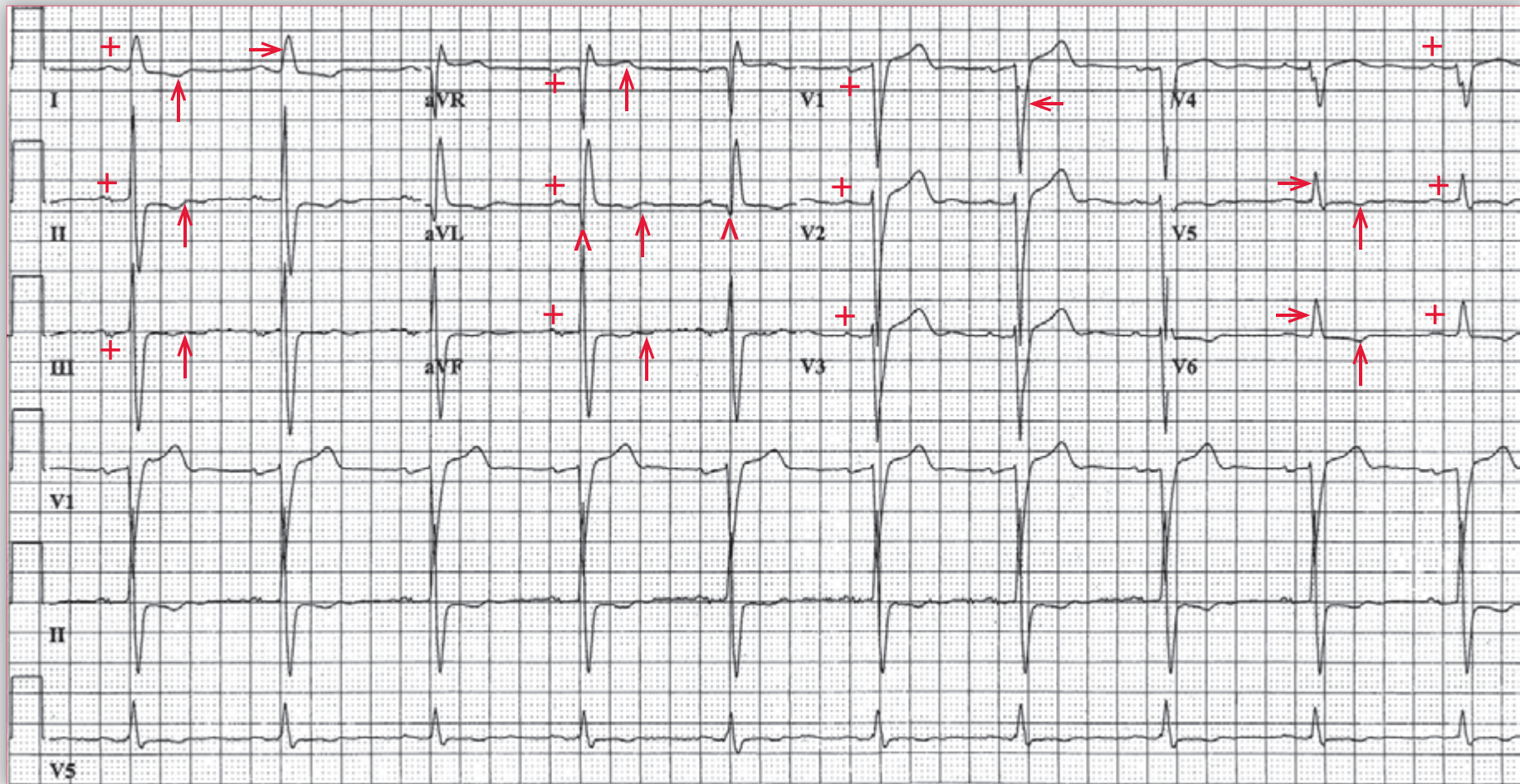
What abnormalities are noted for ECG 4A?

What abnormalities are now noted on ECG 4B, and how may they relate to the first tracing (ECG 4A)?

ECG 4B



Podrid's Real-World ECGs



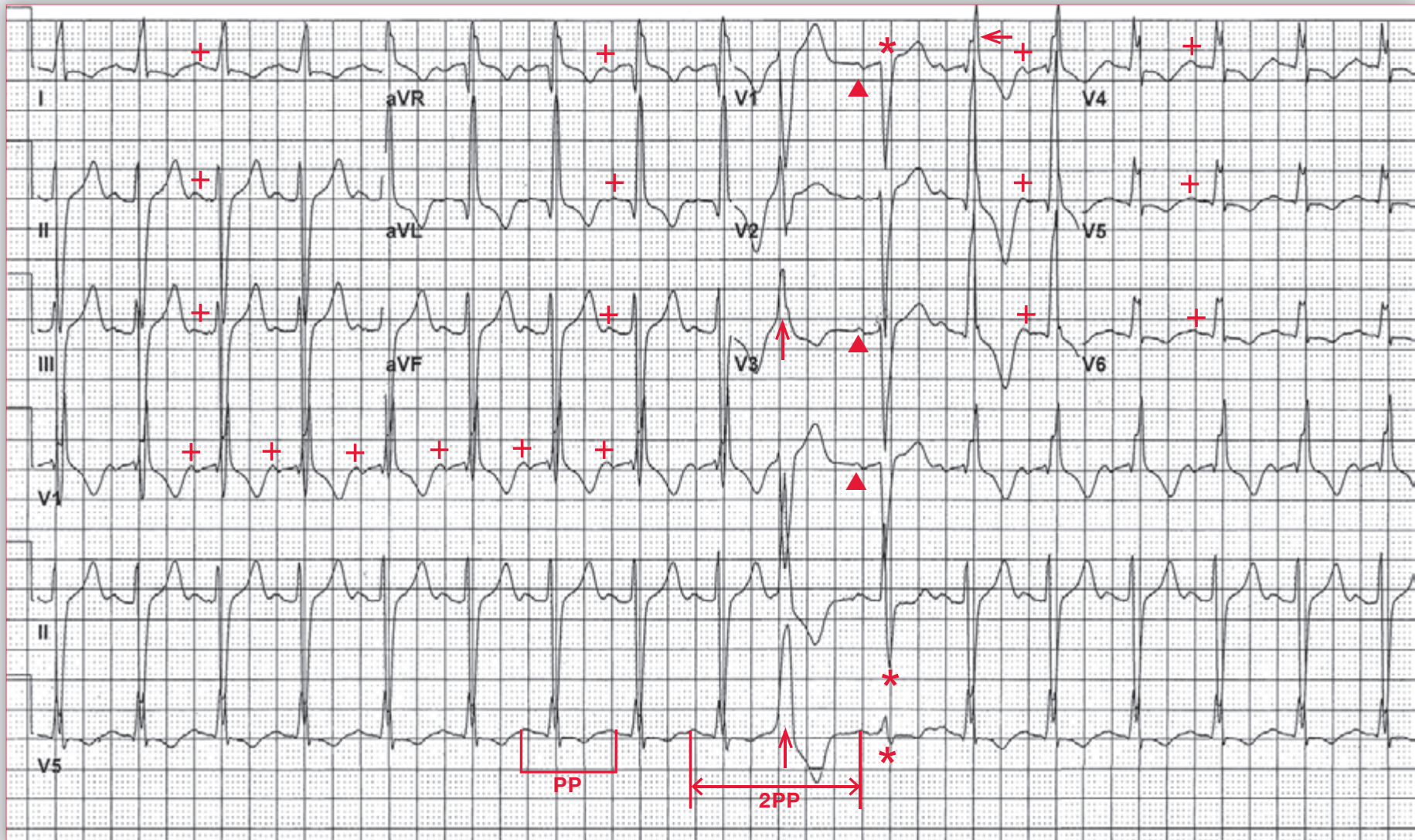
ECG 4A Analysis: Normal sinus rhythm, intraventricular conduction delay, nonspecific ST-T wave abnormalities

In ECG 4A, there is a regular rhythm at a rate of 60 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus rhythm. The QRS complex duration is increased (0.12 sec). Although the QRS morphology resembles a left bundle branch block (broad R wave in lead I and V5–V6 [→] and QS complex [←] in lead V1, there is a septal Q wave in lead aVL (^). There is a septal branch of the left bundle that innervates the intraventricular septum; the direction of this initial activation is from left to right, producing a

small R wave in lead V1 and small septal Q waves in leads I, aVL, and V5–V6. With a left bundle branch block, this branch is also blocked and not active. Therefore, there are no septal Q waves seen with a left bundle branch block. Hence the presence of septal Q waves indicates that this is not a left bundle branch block, but rather is an intraventricular conduction delay. The axis is normal, approximately 0° (QRS positive in lead I and biphasic in lead aVF). The QT/QTc intervals are normal (440/440 msec). Also present are diffuse ST-T wave changes (↑).

continues

Podrid's Real-World ECGs



ECG 4B Analysis: Sinus tachycardia, rate-related left anterior fascicular block, rate-related right bundle branch block, premature ventricular complex with compensatory pause

ECG 4B is from the same patient as ECG 4A. There is a regular rhythm at a rate of 100 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia.

The QRS complex duration is increased (0.14 sec) and the morphology resembles a right bundle branch block (RSR' in lead V1 [←]) although broad S waves are not seen in leads I or V5–V6). The axis is extremely leftward between -30° and -90° (positive QRS in lead I and negative in leads II and aVF). There are two causes for an extreme left axis, *ie*, an inferior wall myocardial infarction in which there is an initial Q wave in leads II and aVF and a left anterior fascicular block in which the QRS complex has an rS morphology, as is seen in this case. Hence this is left anterior fascicular block. The QT/QTc intervals are slightly prolonged (360/460 msec) but normal when the prolonged QRS complex duration is considered (320/410 msec).

The tenth QRS complex is premature (↑); it is wide, it has an abnormal morphology, and there is no P wave before it. Hence this is a premature ventricular complex. There is a compensatory pause (↔) after this premature complex (*ie*, the PP interval around the premature complex is twice the underlying sinus PP interval [⊐]). The QRS complex following the premature complex (*) and after the pause (*ie*, at a slower rate) has an increased duration (0.12 sec), resembling a left bundle branch block, and the axis is less leftward (*ie*, QRS complex is positive in lead II). The QRS complex morphology, duration, and axis are identical to that seen in ECG 4A. There is a P wave before this complex (▲) that is identical to the other sinus P waves (+). Therefore, with the heart rate of 100 bpm, there is a rate-related right bundle branch block and left anterior fascicular block. Although a rate-related bundle branch block is more commonly seen, there can also be rate-related fascicular blocks. ■

Notes

A 72-year-old woman with a history of prior inferior myocardial infarction but preserved LV function presents with lethargy and decrease in exertional capacity. She states that for the past several months, she has noted difficulty completing her usual daily 2-mile walks, and in general has felt easily fatigued with daily activities.

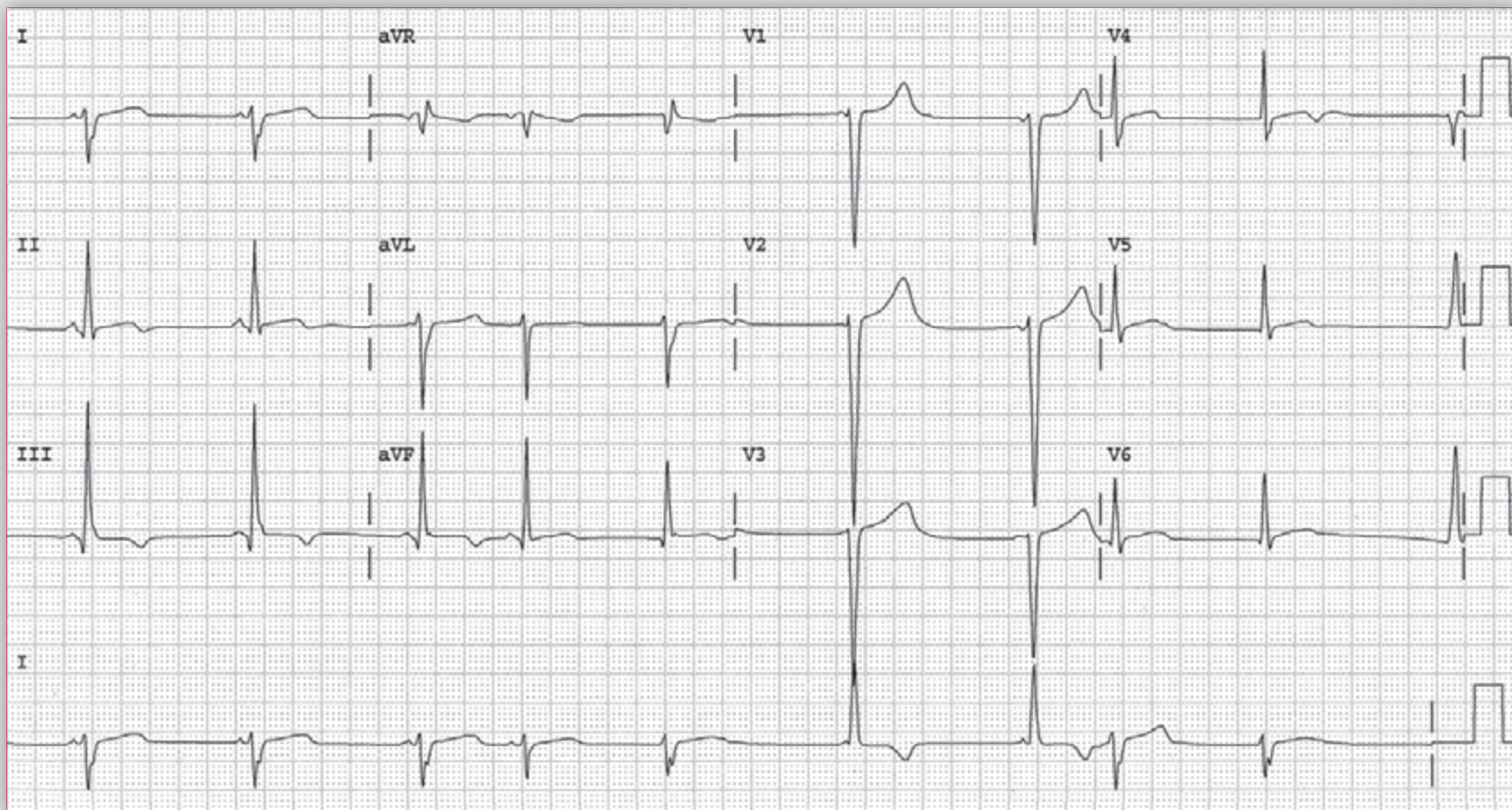
She denies recent illnesses, fevers, signs of gross or occult bleeding, and mood disturbances. She has not had any recent social stressors. There have not been any changes in her medical regimen.

On exam, she appears well, younger than stated age. Her heart rate is irregular at 60 bpm,

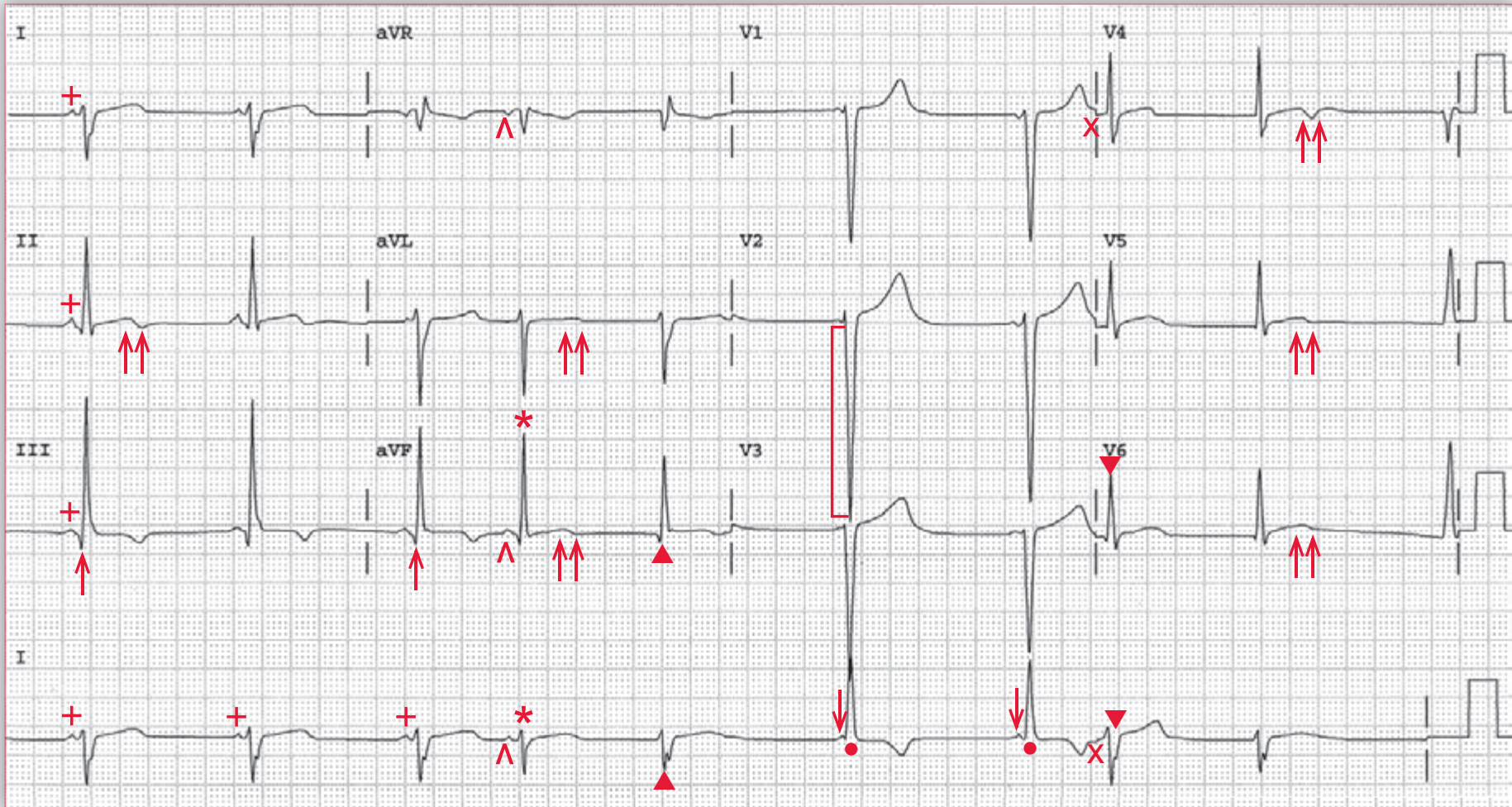
and her blood pressure is 124/62. Her physical exam is unremarkable.

An ECG is obtained as part of her evaluation.

What abnormalities are noted that may explain her symptoms?



Podrid's Real-World ECGs



ECG 5 Analysis: Sinus bradycardia, short PR interval, right axis deviation consistent with left posterior fascicular block, premature atrial complex, escape junctional complexes, nonspecific ST-T wave changes, inferior wall myocardial infarction

There is an irregular rhythm with an average rate of 60 bpm. The first three QRS complexes are regular at a rate of 50 bpm. There is a P wave (+) in front of each QRS complex with a stable PR interval (0.12 sec). Hence these are sinus complexes with a short PR interval. The QRS complex duration is normal (0.10 sec) and there is a rightward axis with an rS morphology in lead I between +90° and +180° (negative QRS complex in lead I and positive in lead aVF). As there are no other ECG abnormalities associated with any other etiologies for a rightward axis, *ie*, lateral wall myocardial infarction, right ventricular hypertrophy, R-L arm lead switch, dextrocardia, or Wolff-Parkinson-White, this is termed a left posterior fascicular block. There are Q waves seen in leads III and aVF (↑). Although they are not deep, they are wide (at lead 0.04 sec) consistent with an old or chronic inferior wall myocardial infarction. The QT/QTc intervals are normal (480/440 msec).

The fourth QRS complex is premature (*), and there is a P wave (^) before it with a slightly different morphology compared to the first three sinus P waves. This is a premature atrial complex and the QRS morphology is identical to that of the first three QRS complexes. The fifth QRS complex (▲) (at a rate of 60 bpm) has the same morphology, but there is no P wave before this QRS complex. Hence it is a junctional complex. The sixth and seventh QRS complexes (●) are positive in lead I and hence a left posterior fascicular block is no longer present; it cannot be established if the axis is either normal or leftward, as this complex is not seen in aVF. The QRS complex duration is the

same as the other QRS complexes (0.10 sec). There is an increased voltage (*ie*, S wave in V2 = 34 mm) ([], consistent with left ventricular hypertrophy (*ie*, an S wave or R wave in any precordial lead > 25 mm). Although there are P waves seen before these QRS complexes (↓), the PR intervals are very short and different for each of these the two complexes. Hence the P waves are nonconducted and the QRS complexes are junctional. They are at a rate of 48 bpm. The eighth QRS complex is premature (▼) and there is a P wave (x) before it. This is a premature atrial complex and the QRS complex again has a rightward axis or left posterior fascicular block, similar to the axis of the first 4 QRS complexes. The last (ninth) QRS complex has no P wave before it and the QRS complex is identical to the first five, *ie*, there is a rightward axis or left posterior fascicular block.

It can be noted that the RR intervals are shorter whenever the QRS complexes have a left posterior fascicular block (rightward axis). Hence this represents a rate-related left posterior fascicular block. Although rate-related bundle branch block is more commonly seen, there can also be rate-related fascicular blocks.

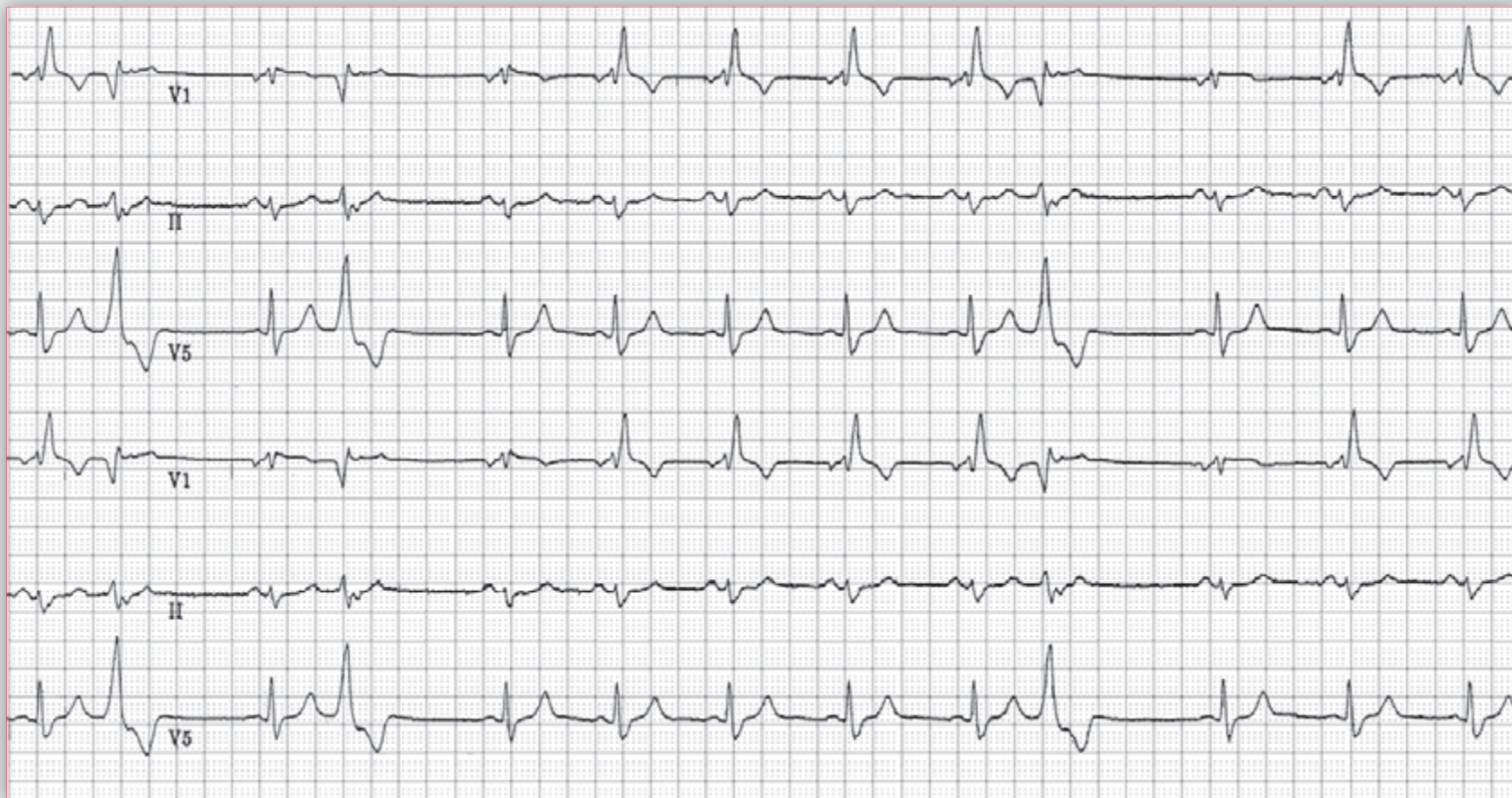
Also noted are T-wave abnormalities in leads II, aVL, aVF, and V4–V6 (↑↑). It is likely that her symptoms are the result of sinus bradycardia and an intermittent slow escape junctional rhythm and also failure to increase her heart rate with exercise, *ie*, chronotropic incompetence. ■

Notes

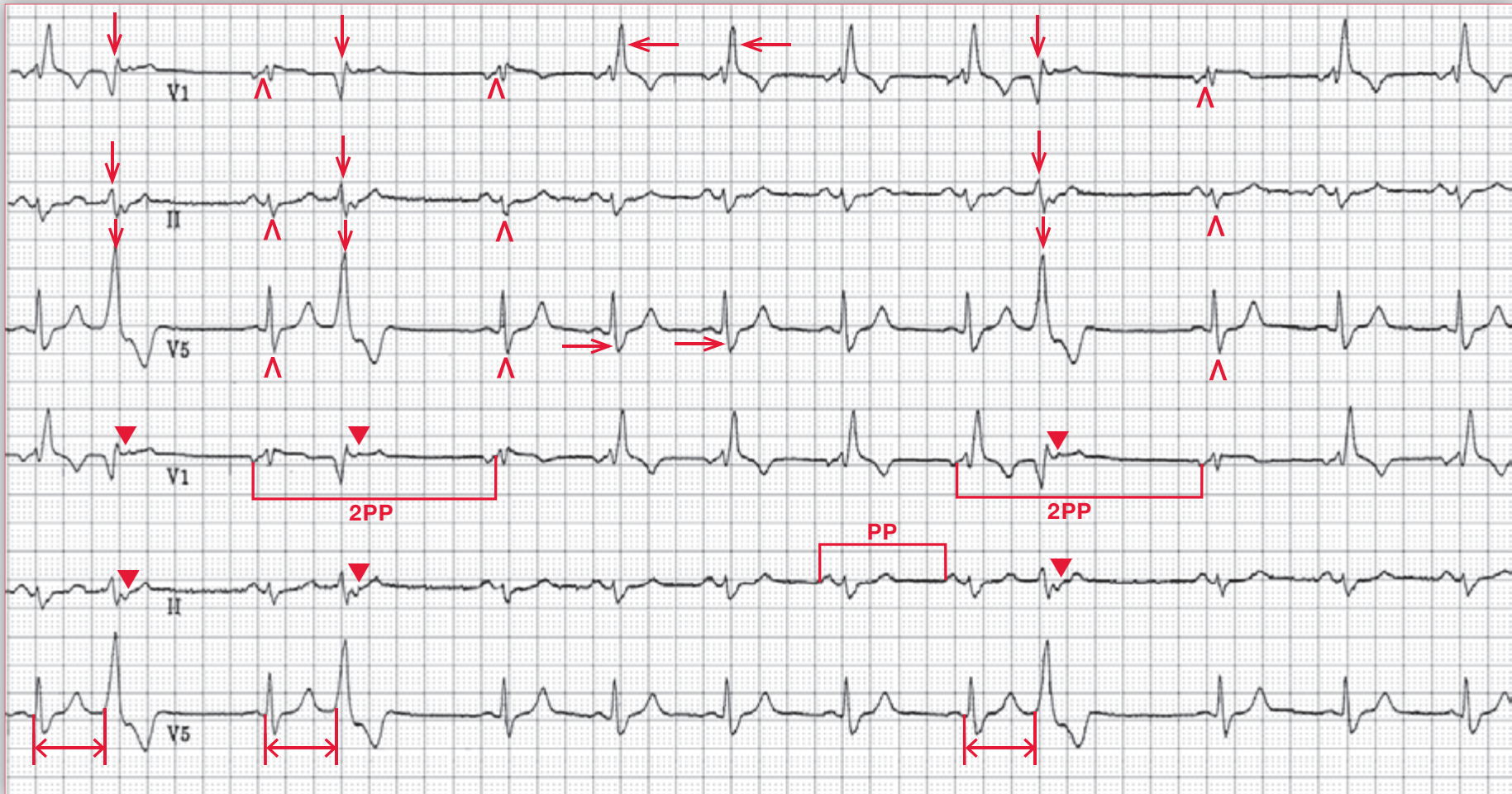
A 44-year-old man presents to his primary care physician with palpitations. He noted the onset several weeks ago. The symptoms last for hours, coming and going without warning. He has been otherwise well and an extensive review of symptoms is unremarkable.

As part of his evaluation, an ECG is obtained.

What abnormalities are noted that may explain his symptoms?



Podrid's Real-World ECGs



ECG 6 Analysis: Normal sinus rhythm, rate-related right bundle branch block morphology, premature ventricular complexes with compensatory pause

The ECG is a series of rhythm strips. The rhythm is regularly irregular as a result of three premature QRS complexes (complexes 2, 4, and 10) (↓) that are followed by a pause. The rest of the QRS complexes are regular at a rate of 76 bpm. All but the third, fifth, and eleventh QRS complexes (^) that follow the premature QRS complexes have a QRS complex duration that is increased (0.14 sec) and a morphology that is consistent with a right bundle branch block, *ie*, RSR' in lead V1 (←) and a prominent widened terminal S wave in V5 (→). The QT/QTc intervals are slightly prolonged (400/450 msec) but are normal when the widened QRS complex duration is considered (360/400 msec).

The three premature QRS complexes (↓) are also wide, but have a morphology that is different from the other QRS complexes. In addition, they are not preceded by a P wave. Hence these are premature ventricular complexes. There is a fixed relationship between each of the premature ventricular complexes (↔) and the QRS complexes that precede them (*ie*, fixed coupling interval). After each premature complex, there is a retrograde P wave (▼) and a pause. The PP interval surrounding the pause (⊐) is equal to two sinus (PP) intervals (⊐) and is a full compensatory pause. Hence this is a normal sinus rhythm with frequent premature ventricular complexes.

It is noted that the QRS complex after each pause (^) is narrow (QRS duration = 0.08 sec) and it does not have a right bundle branch block morphology. Hence there is a rate-related right bundle branch block present, *ie*, the QRS complex has a right bundle branch block morphology when the rate is 76 bpm, but the QRS complex is normal when the rate is 50 bpm (*ie*, the rate associated with the post premature complex pause).

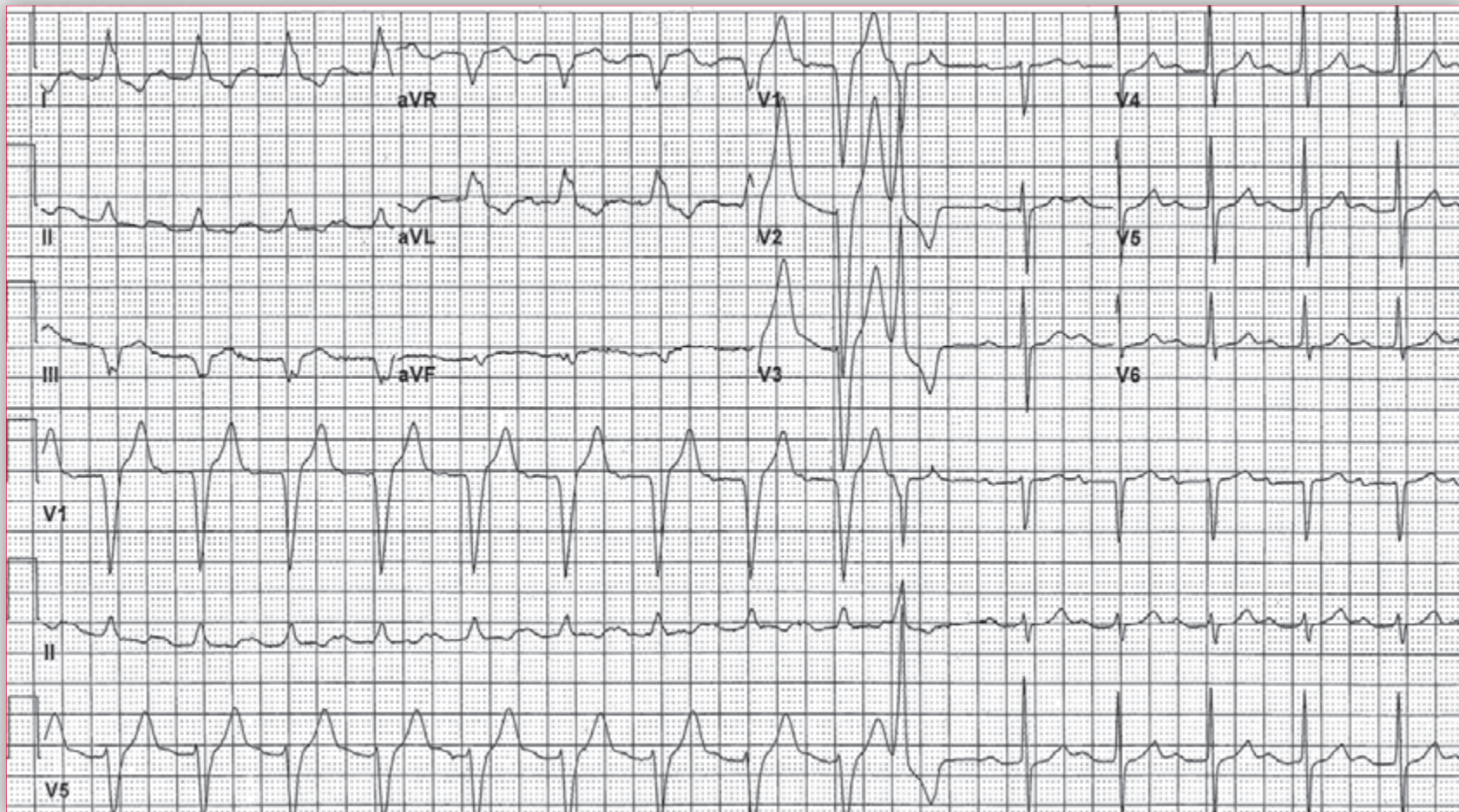
Although rate-related bundles are typically considered to be associated with faster heart rate, a rate-related bundle may occur whenever there is an increase in heart rate, even if the heart rate is not fast, and resolving when the heart rate slows. For example, a right bundle branch block may be present at a rate of > 60 bpm, but not at a rate < 60 bpm.

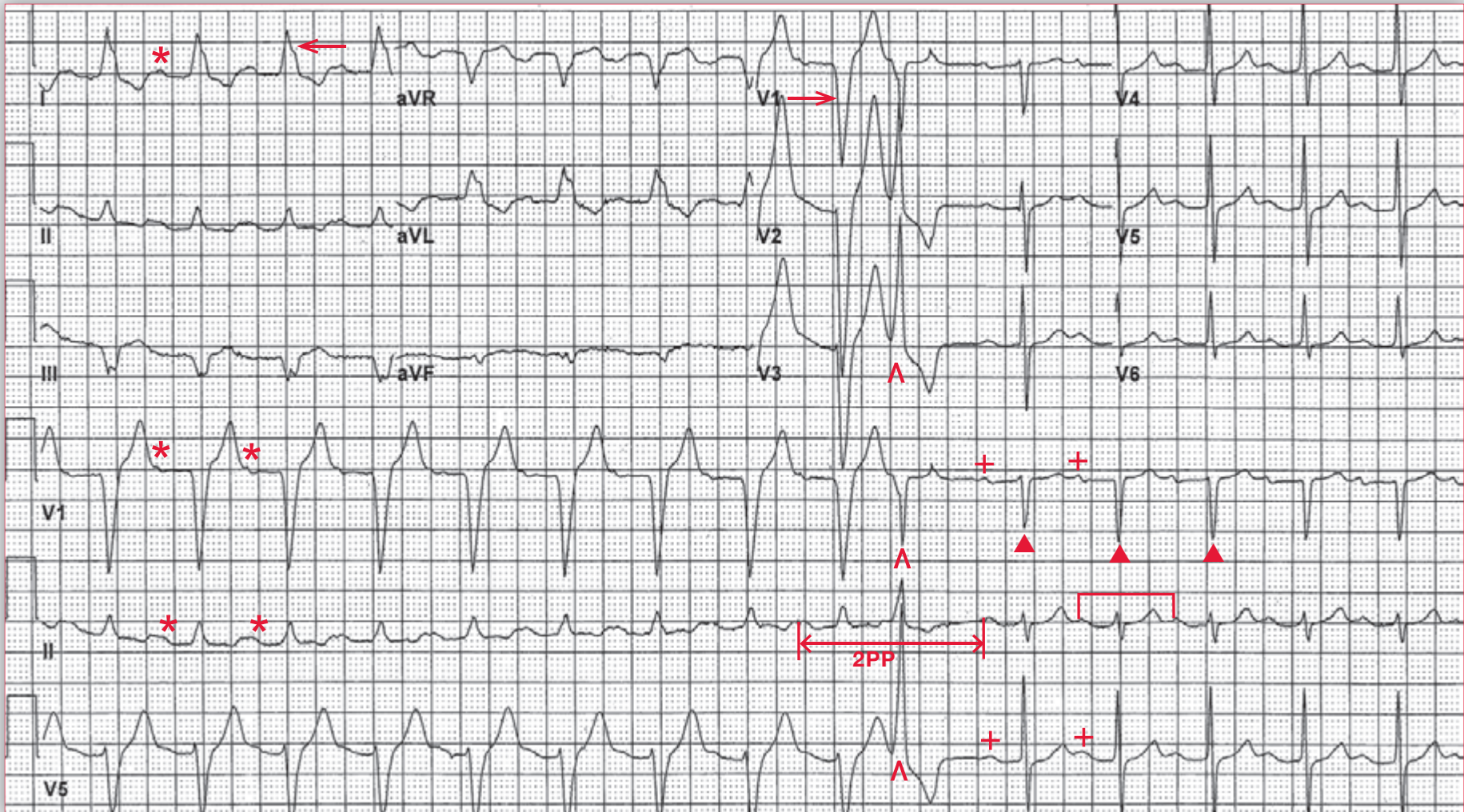
It is likely that the patient's symptoms are related to the premature ventricular complexes and the presence of a compensatory pause. The sensation of palpitations is generally the result of the pause following the premature complex, during which there is continued diastolic filling of the ventricles. With ventricular contraction the increased end diastolic volume results in an increase in inotropy via the Starling effect. This increased myocardial contractility and increased stroke volume causes the sensation of palpitations. Premature ventricular complexes are benign and do not require any specific therapy. The patient should be reassured that the palpitations are the result of a benign arrhythmia. ■

Notes

A cardiologist is auscultating her patient's heart sounds. During the exam, she notes that the S2 split, initially not audible, is suddenly audible after a premature ventricular beat. She obtains an ECG to evaluate.

What features of the ECG explain this physician's physical exam finding?





ECG 7 Analysis: Normal sinus rhythm, rate-related left bundle branch block, premature ventricular complex

The rhythm is relatively regular at a rate of 94 bpm. There is some irregularity due to the tenth QRS complex (^) that is premature, with a long RR interval or pause following it. There is a P wave before each of the QRS complexes (*) (except for the tenth or premature complex) and the PR interval is constant (0.18 sec). Therefore, this is a normal sinus rhythm. The QRS complex duration is increased (0.14 sec) and has a morphology typical for a left bundle branch block, *ie*, there is a tall, broad R wave in lead I (←) and a deep QS complex in lead V1 (→). The axis is leftward (positive QRS complex in leads I and II and negative in lead aVF). The QT/QTc intervals are prolonged (400/500 msec) but are normal when the prolonged QRS complex duration is considered (360/440 msec).

The premature complex has a different morphology and is not preceded by a P wave. Hence this is a premature ventricular complex, which is followed by a compensatory pause, *ie*, the PP interval surrounding the premature ventricular complex (↔) is equal to two sinus (PP) intervals (□).

Following the premature ventricular complex and the pause, there are regular QRS complexes (▲) that are preceded by a P wave (+) with the same morphology and PR interval as seen before the premature ventricular complex. Hence this is still a normal sinus rhythm. However, the QRS complexes are narrow (0.08 sec) and no longer have a left bundle branch block morphology (▲). This is likely due to a slightly

slower sinus rate after the pause (although this is not perceivable on the ECG that is recorded at 25 mm/sec). Hence this represents a rate-related left bundle branch block. It should be pointed out that the rate with and without a bundle branch block may be only very slightly different. Moreover, the rate at which left bundle branch block develops may be different than the rate at which the left bundle branch block resolves. It should be noted that the measured QT interval of these narrow QRS complexes is 360 msec, identical to the QT interval measurement obtained with the complexes showing the bundle when QRS width was considered.

As the second heart sound (S2) is comprised of the closing of the aortic (A2) followed by the pulmonic valves (P2), during left bundle branch block, the normal A2-P2 orientation is altered. In these situations, A2 may superimpose or even come after P2 (P2-A2) due to delayed activation and relaxation of the left ventricle. Given the changes in blood flow across the pulmonic valve during inspiration (specifically negative thoracic pressure during inspiration increasing venous return and increasing pulmonary blood flow), normally the A2-P2 split widens during inspiration. When P2 follows A2, the S2 split disappears or becomes smaller during inspiration (“paradoxical splitting”). In the case presented, left bundle branch block resulted in superimposed A2 and P2. When the sinus rate decreased after the PVC to allow for a normal QRS duration, the normal physiologic splitting of S2 (A2-P2) was restored. ■

Core Case 8

A 67-year-old man presents to hospital with chest pain. He states he was well until 2 weeks prior to presentation when he experienced a nonspecific upper respiratory syndrome. Symptoms resolved completely and he was well until the night prior to presentation, when he noted the indolent onset of a positional chest pain. The pain is described as sharp, without the

quality of pressure or heaviness. The discomfort does not radiate; however, it worsens with deep breathing.

His medical history is notable for CAD and a prior inferior myocardial infarction, peptic ulcer disease, and an upper gastrointestinal bleed in the distant past as well as depression. His medications include aspirin and a statin.

ECG 8A



On exam, his vital signs are notable for tachycardia and mild hypertension. His physical exam, including cardiovascular exam, is unremarkable.

As part of his workup, an ECG is obtained (ECG 8A). On interpretation of the ECG, the emergency room physician urgently pages the cardiologist on call and inquires as to

the need for an emergent coronary angiography.

The patient is treated with aspirin, intravenous β -blockers, intravenous nitrates, and heparin. A repeat ECG is obtained (ECG 8B). Upon interpretation of this ECG, the emergency physician calls the cardiologist once again and wonders if the emergent cardiac catheterization can be deferred.

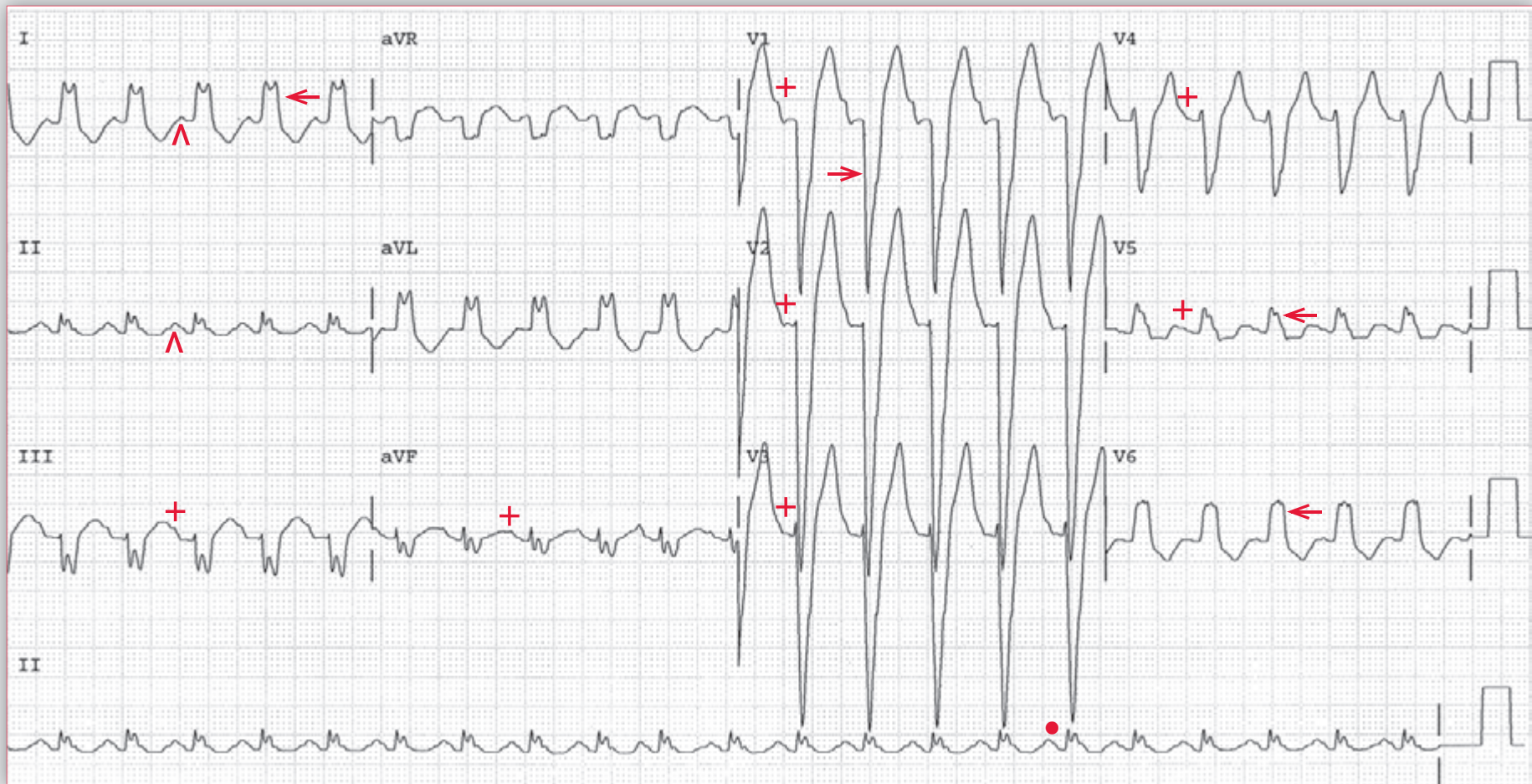
What abnormalities on the ECG 8A prompted the physician's concern?

What findings on ECG 8A prompted this re-assessment?

ECG 8B



Podrid's Real-World ECGs



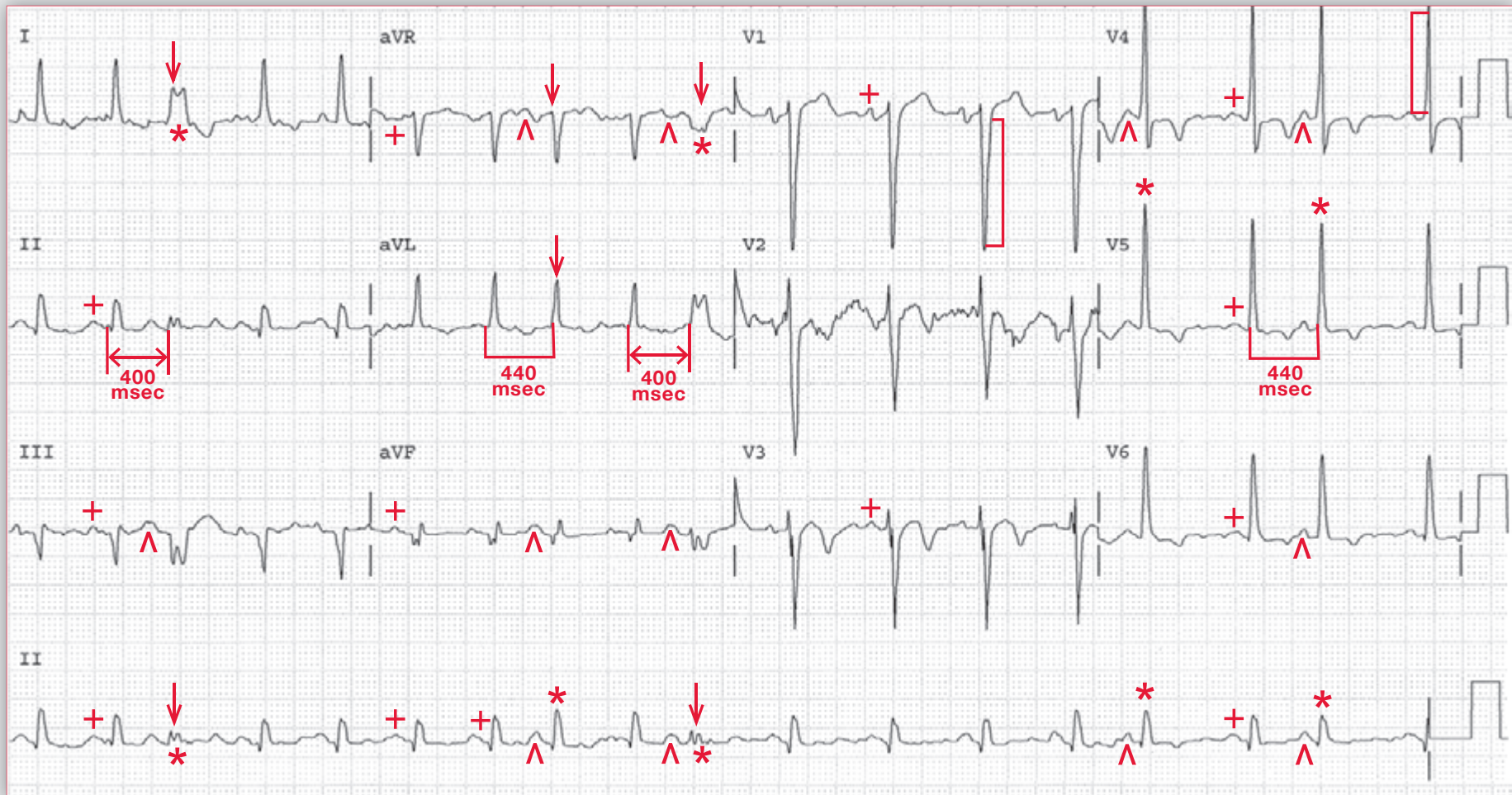
ECG 8A Analysis: Sinus tachycardia, left bundle branch block

ECG 8A shows a regular rhythm at a rate of 130 bpm. There are P waves (+) before each QRS complex, seen as positive waveforms on the downslope of the T wave in leads III, aVF, and V1–V5). A distinct P wave can be seen in leads I and II (^). It should be remembered that the T wave should have a smooth upstroke and downstroke; any notching, bump or other irregularity on the T wave suggest a superimposed P wave. The PR interval is stable (0.16 sec). The P waves are positive in leads I, II, aVF, and V4–V5. Hence this is a sinus tachycardia.

The QRS complex duration is prolonged (0.16 sec), and there is a typical left bundle branch block morphology (broad R wave in leads I and V5–V6 [←] and QS complex in lead V1 [→]). The QT/QTc intervals are prolonged (360/530 msec), but are normal when the prolonged QRS complex duration is considered (300/420 msec). Therefore, this is a sinus tachycardia with a left bundle branch block. Without careful inspection of the ECG and the identification of P waves before each QRS complex, the ECG shows a wide complex tachycardia and this would lead to a concern that the rhythm was ventricular tachycardia.

continues

Podrid's Real-World ECGs



ECG 8B Analysis: Normal sinus rhythm, premature atrial complexes with left bundle branch block (rate-related), old inferior wall myocardial infarction, left ventricular hypertrophy

ECG 8B shows that the rhythm is primarily regular at a rate of 92 bpm, although there are some QRS complexes that are premature, *ie*, the third, eighth, tenth, fifteenth, and seventeenth (*). There is a P wave (+) before each of the regular QRS complexes with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6; hence this is a normal sinus rhythm. The QRS complex duration is normal (0.10 sec) and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (340/420 msec). The QRS voltage is increased with an R wave = 22 mm in lead V4 ([]) and S wave = 24 mm in lead V1 ([]), meeting criteria for left ventricular hypertrophy (*ie*, 46 mm in total).

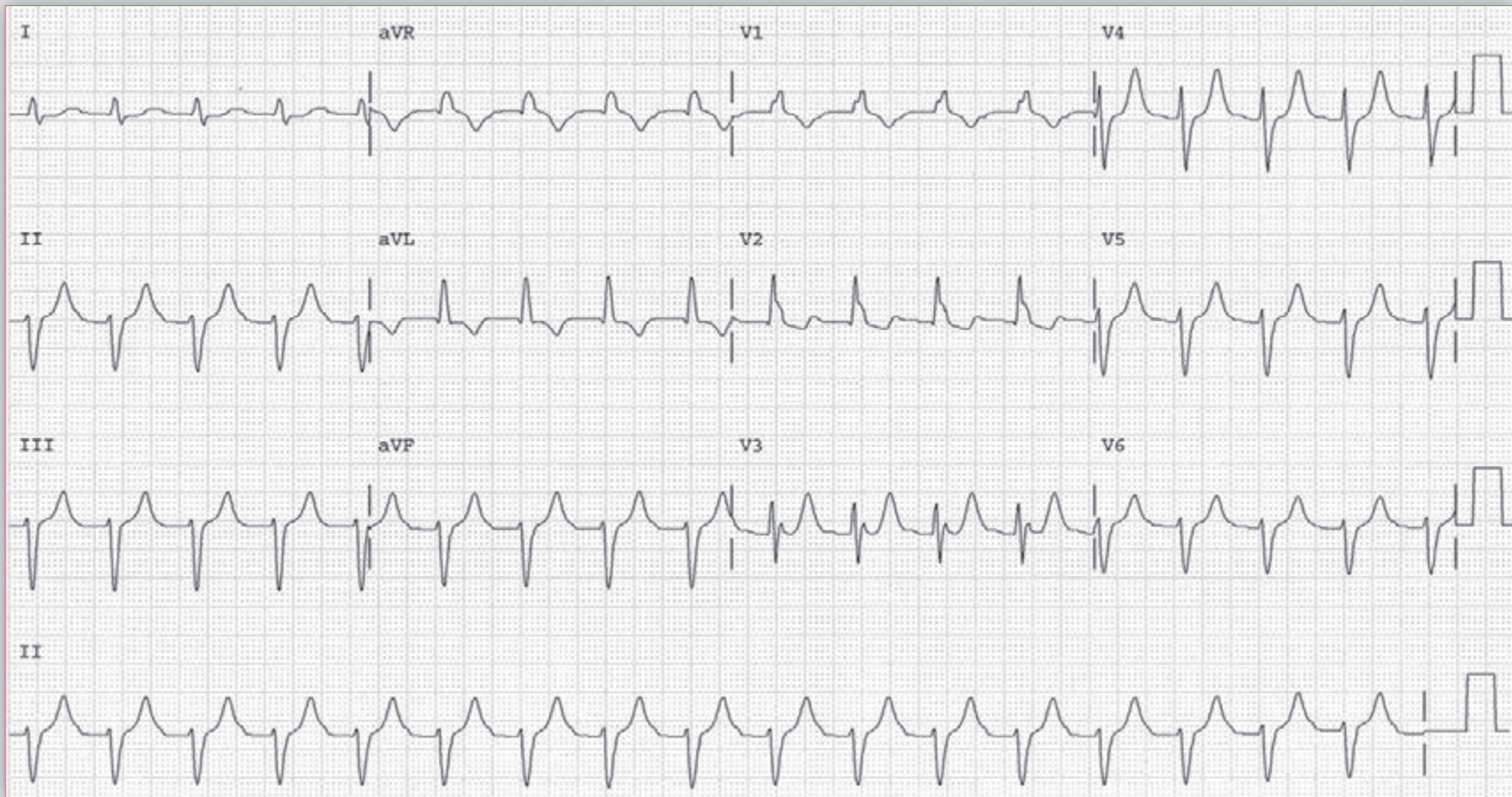
The third, eighth, tenth, fifteenth, and seventeenth QRS complexes are premature (*). They are preceded by a P wave (^) and all except the third and tenth have the same QRS morphology as the sinus complexes. Hence these are premature atrial complexes.

Complexes 3 and 10 (↓) have a shorter coupling interval (*ie*, RR interval = 400 msec) (↔) compared to the other premature atrial complexes (RR interval 440 msec) (⊥) and the QRS morphology is different. The morphology of complexes three and ten is identical to the QRS morphology seen in ECG 9A, *ie*, they have a left bundle branch block morphology. Hence these premature complexes have a rate-related left bundle branch block. As these premature complexes have the same morphology as the QRS complexes in ECG 8A, this confirms that ECG 8A shows sinus tachycardia with a rate-related left bundle branch block. Although there was likely an initial concern that the ECG showed ventricular tachycardia, the finding of a P wave before each QRS complex and the fact that the aberrated premature atrial complexes have exactly the same morphology as the wide QRS complex in ECG 8A confirms the fact that the rhythm is sinus tachycardia with a rate-related left bundle branch block. ■

Core Case 9

A 42-year-old man with a prior history of inferior myocardial infarction presents with several weeks of spontaneous palpitations. He states that his heart “races” without warning. He denies any other symptoms and cannot point to any triggers. The sensation terminates spontaneously minutes later. Of note, his primary doctor

ECG 9A

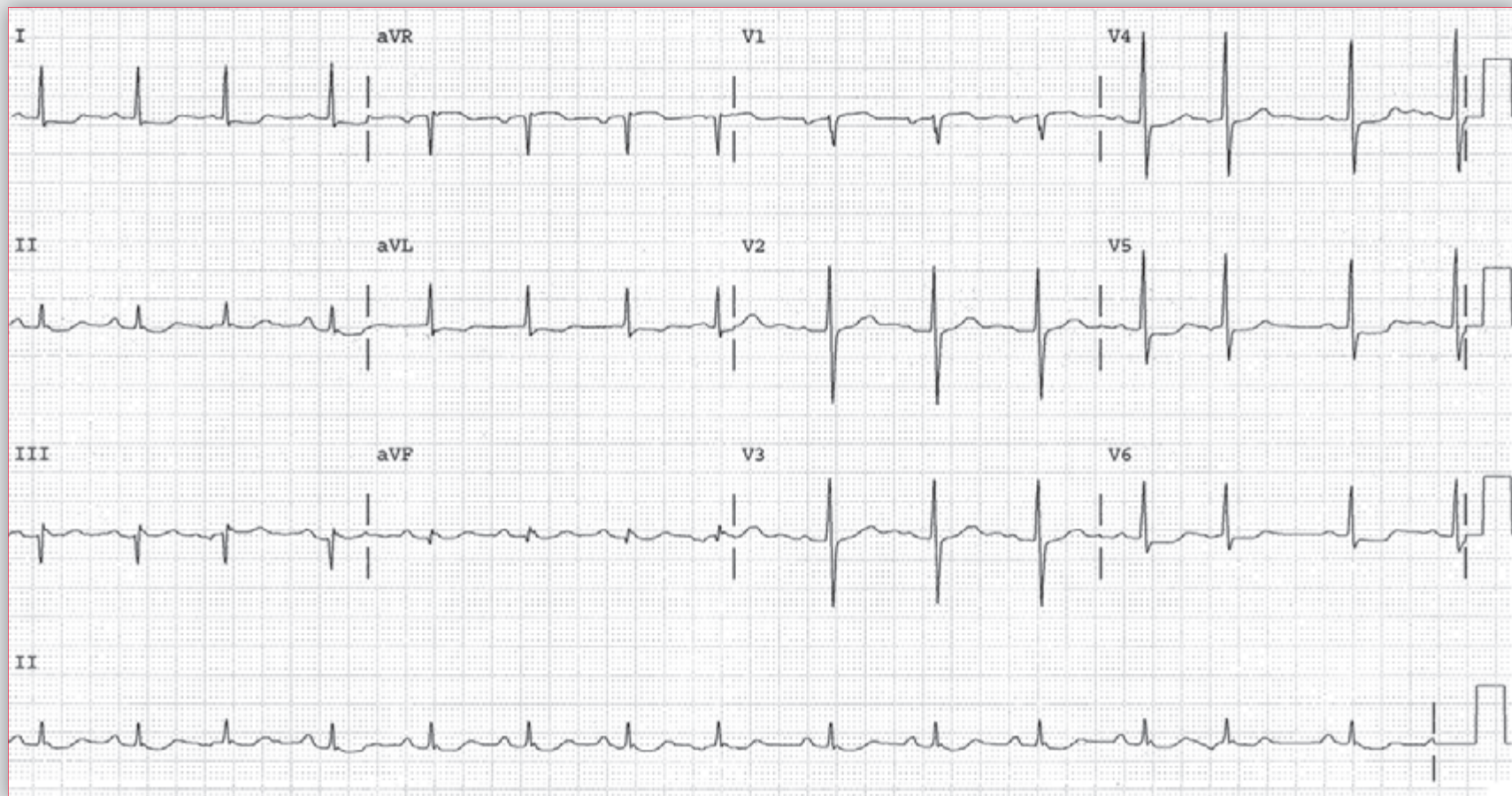


recently decreased his β -blocker dose because of erectile dysfunction. He has not otherwise had any changes in his medical regimen.

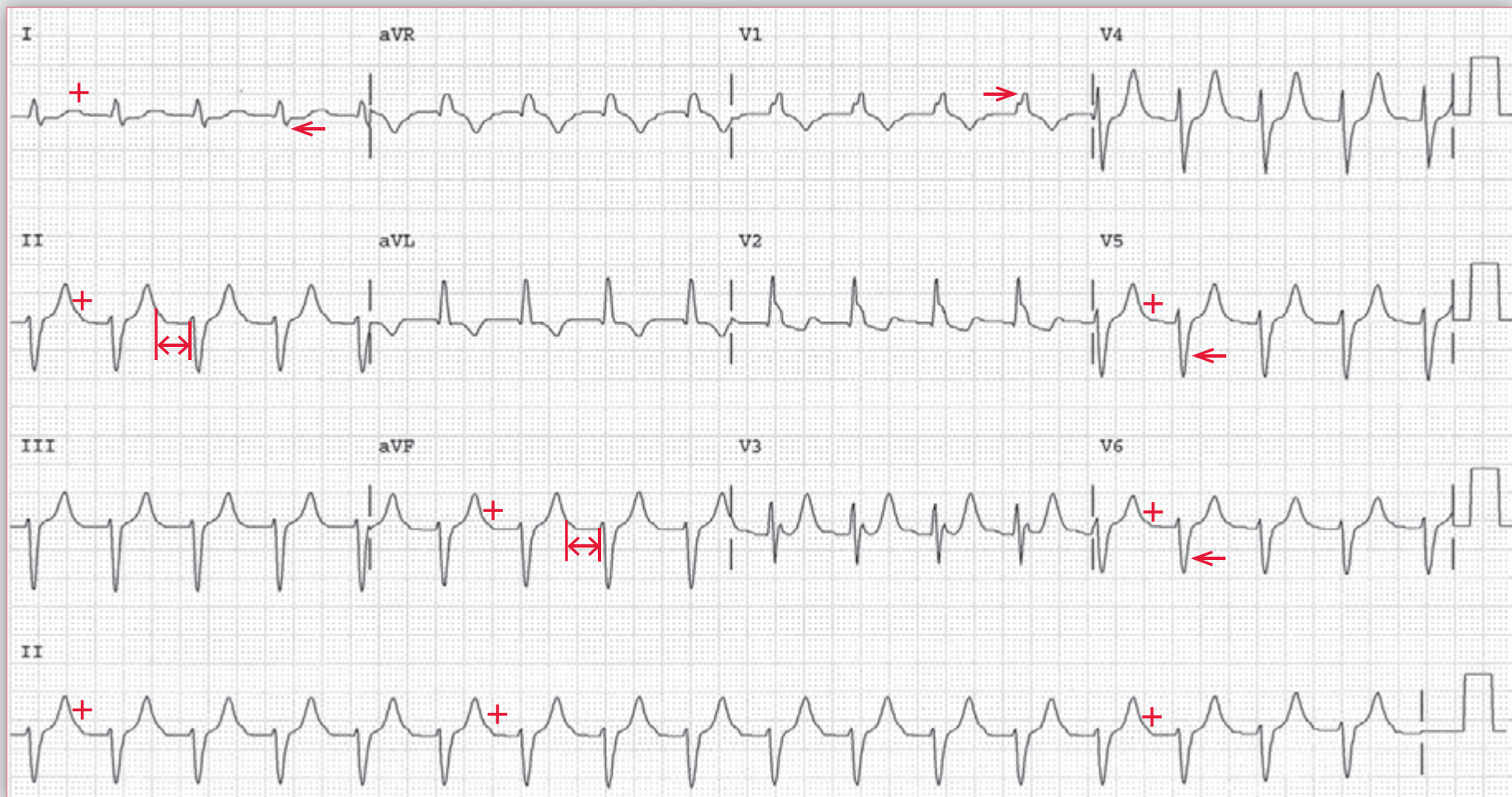
On exam, a rapid radial pulse is noted. An ECG is obtained (ECG 9A). Several minutes later, the radial pulse returns to normal and a follow-up ECG is obtained (ECG 9B).

What abnormalities are noted on the ECGs, and what diagnosis is suggested?

ECG 9B



Podrid's Real-World ECGs



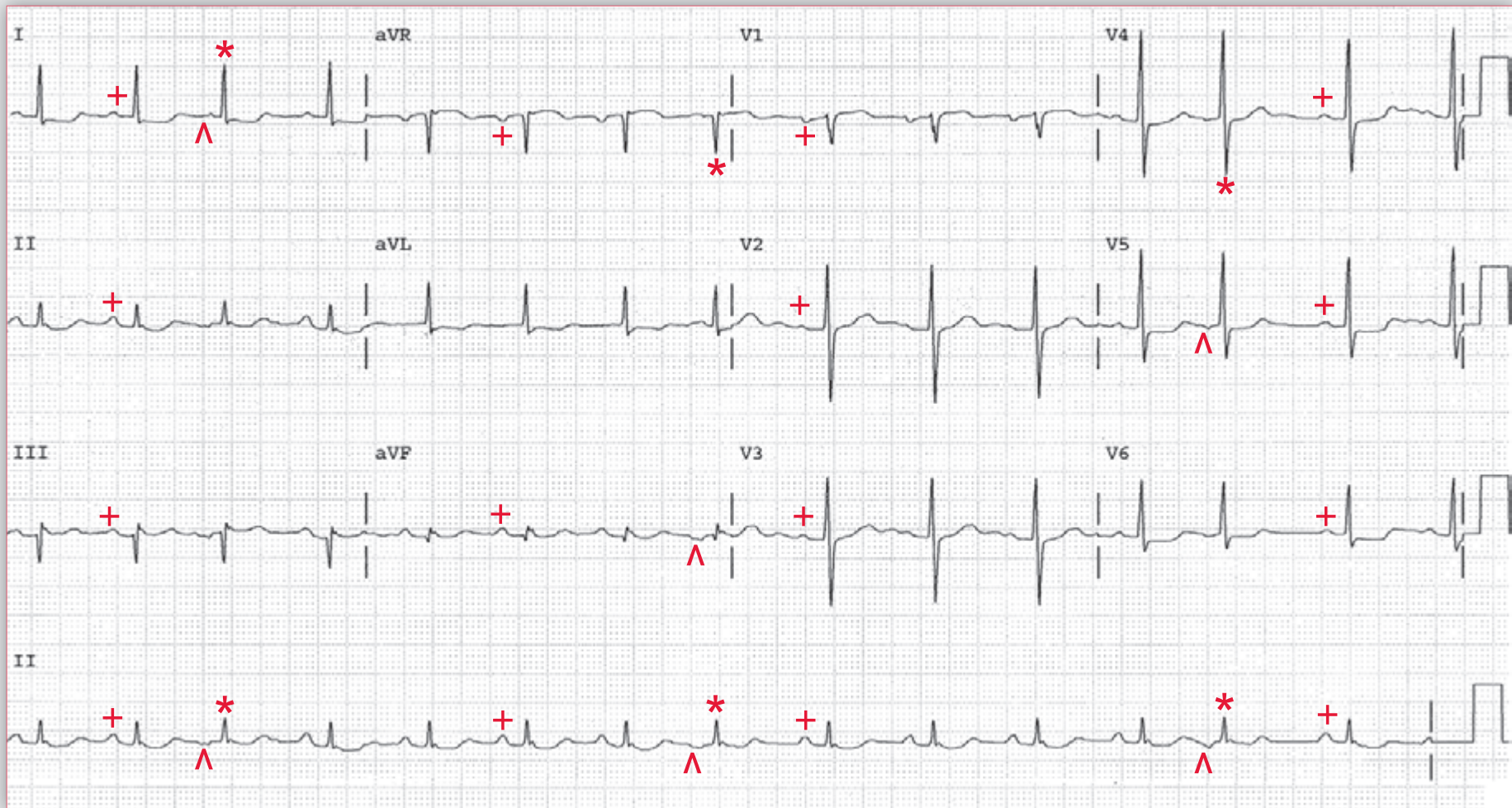
ECG 9A Analysis: Short-RP tachycardia due to an atrial tachycardia, rate-related left anterior fascicular lock, rate-related right bundle branch block

ECG 9A shows a regular rhythm at a rate of 110 bpm. There is a P wave before each QRS complex best seen at the end of the T wave in leads I, II, aVF, and V5–V6 (+). It should be remembered that the normal T wave has a smooth upstroke and downstroke; any bumps, notches, or other irregularity on the T wave is strongly suggestive of a superimposed P wave. There is a stable PR interval (0.28 sec) (\leftrightarrow). The P wave is positive in leads II, aVF, and V6. Thus, this appears to be a sinus tachycardia with a first-degree AV block, or AV conduction delay.

The QRS complex duration is increased (0.14 sec) and the morphology is that of a right bundle branch block with a broad R wave in lead V1 (\rightarrow) and a broad S wave in leads I and V5–V6 (\leftarrow). In addition, the axis is markedly leftward between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF). As the QRS complex has a rS morphology, this is a left anterior fascicular block. The QT/QTc intervals are slightly prolonged (360/490 msec), but normal when the prolonged QRS complex duration is considered (320/430 msec).

continues

Podrid's Real-World ECGs



ECG 9B Analysis: Normal sinus rhythm, premature atrial complexes

ECG 9B is from the same patient as ECG 9A. There is basically a regular rhythm at a rate of 88 bpm. There is some irregularity as the third, eighth, and thirteenth QRS complexes are premature (*). There is a P wave before each regular QRS complex (+) with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4–V6; hence this is a normal sinus rhythm. There is a P wave also seen before the premature QRS complexes (^), but the P-wave morphology is different from the sinus P wave; these are, therefore, premature atrial complexes.

It should be noted that although the rhythm in ECG 9A appears to be sinus tachycardia, the PR interval is longer during this tachycardia (*ie*, 0.28 sec) compared to the PR interval seen in this ECG (*ie*, 0.18 sec) in which there is a sinus rhythm at a slower rate of 88 bpm. Sinus tachycardia, which is due to increased catecholamines or sympathetic tone, is associated with a shortening of the PR interval because of an increase in the conduction velocity through the AV node. In this case, the PR interval is longer at a faster heart rate and shorter at the slower heart rate. This is not seen with sinus tachycardia, and therefore the rhythm

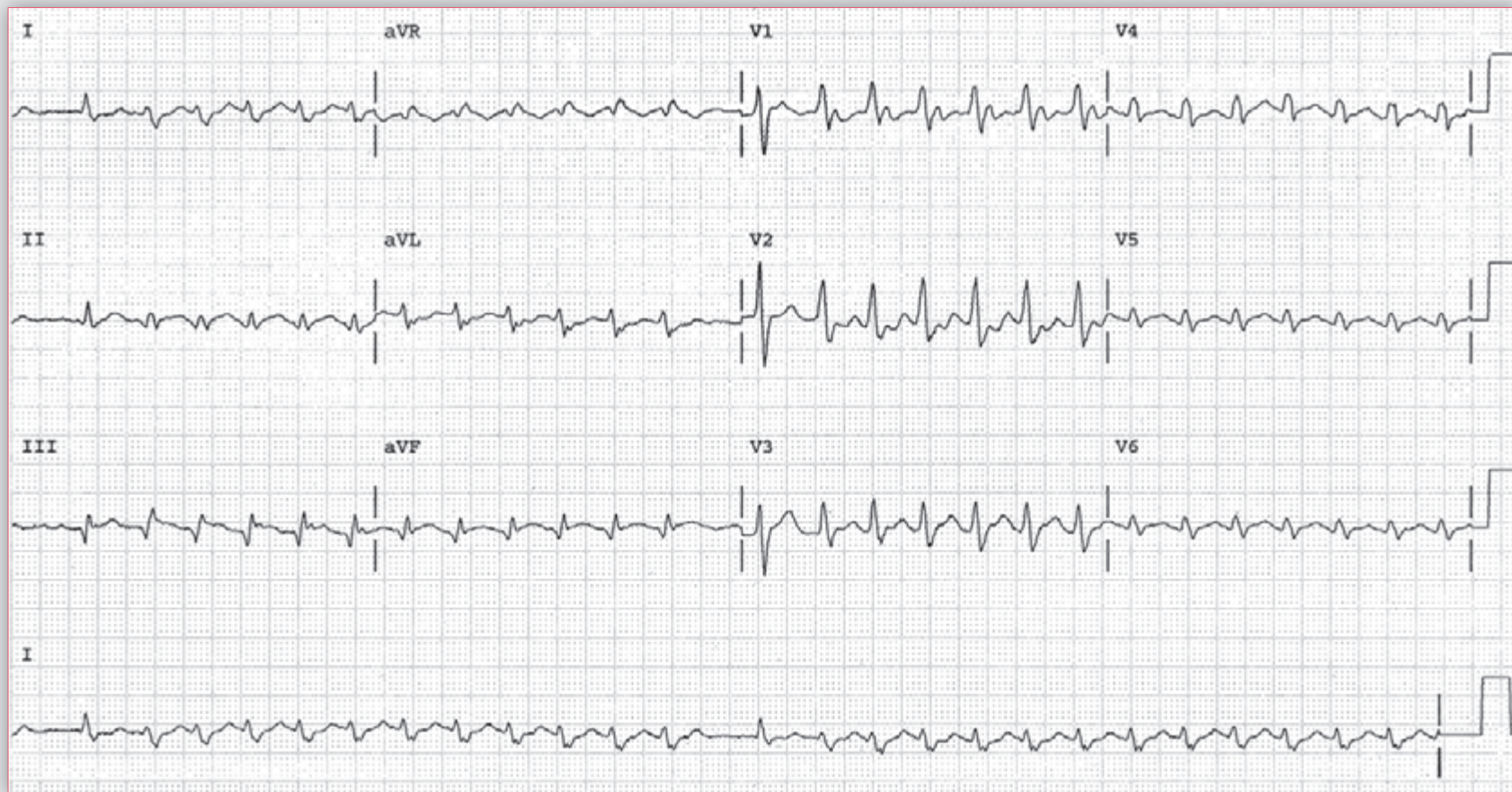
in ECG 9A is not sinus tachycardia, but rather is an atrial tachycardia. Atrial tachycardia, which is not generally mediated by catecholamines, is often associated with a PR interval that is longer than that seen during sinus rhythm, a result of decremental AV nodal conduction (*ie*, conduction through the AV node slows when it is stimulated at a faster rate in the absence of catecholamines). As the P-wave axis is consistent with a sinus mechanism, the atrial focus is in the upper part of the right atrium, near the sinus node.

The QRS complex duration is normal (0.08 sec) and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QRS morphology is normal. The QT/QTc intervals are normal (360/440 msec). There are diffuse nonspecific ST-T wave changes noted. As the conduction abnormalities seen in ECG 9A developed when the ventricular rate was increased, this is a rate-related right bundle branch block and a rate-related left anterior fascicular block (*ie*, bifascicular block). ■

Core Case 10

A 78-year-old woman presents to the emergency department with persistent palpitations for the past several hours. She complains of associated dyspnea. Her vital signs reveal hemodynamic stability but an elevated heart rate. Her oxygen saturation is normal. Her exam is

ECG 10A



notable for a rapid and regular radial pulse and an elevated JVP. Her lungs sounds are normal.

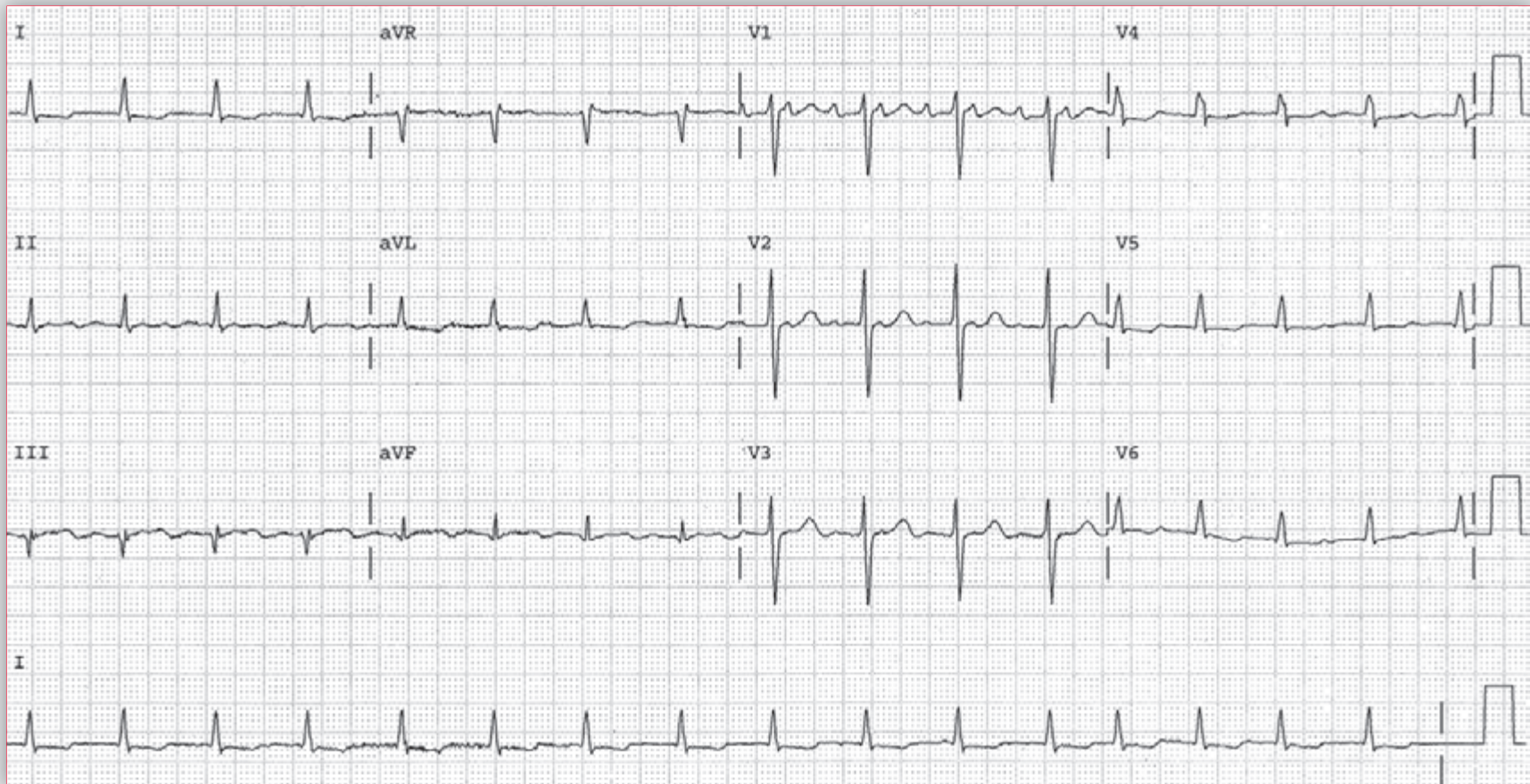
Upon seeing this ECG, the attending physician performs carotid sinus massage and obtains a follow-up ECG.

An ECG is obtained (ECG 10A).

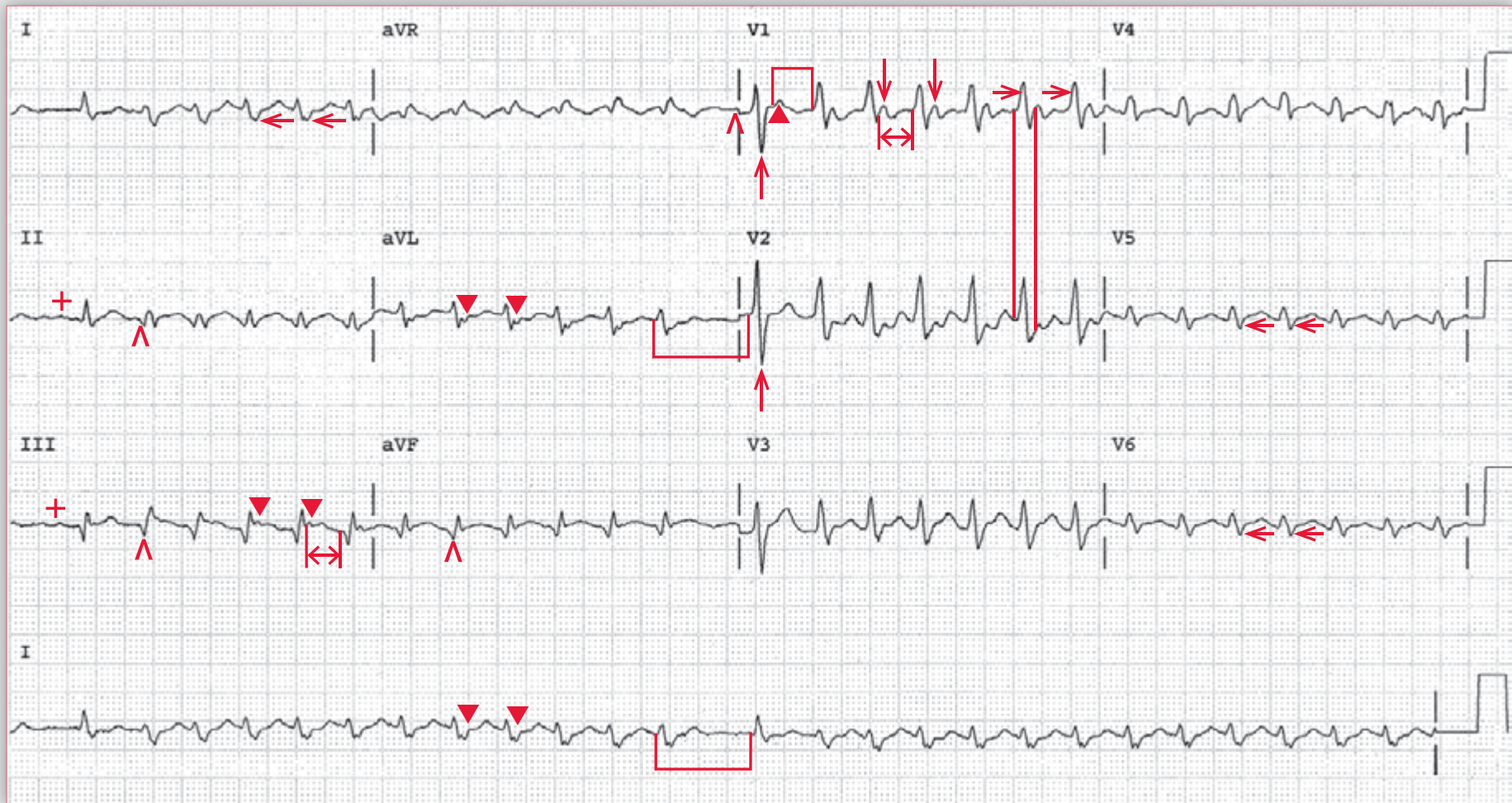
What is your interpretation?

What is the patient's arrhythmic diagnosis?

ECG 10B



Podrid's Real-World ECGs



ECG 10A Analysis: Short-RP tachycardia due to atrial tachycardia with 1:1 conduction, rate-related right bundle branch block, right axis deviation due to left posterior fascicular block, low voltage, possible old inferior wall myocardial infarction

ECG 10A shows that the first QRS complex has a normal duration (0.08 sec) and morphology with a P wave (+) before it and a PR interval of 0.16 second. This is a sinus complex. Following this, there are 11 beats of a regular rhythm at a rate of 180 bpm. The QRS complex duration is increased (0.14 sec) and there is a right bundle branch block morphology (RSR' in V1 [→] and broad S waves in leads I and V5–V6 [←]). The axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). In the presence of a right bundle branch block, there is a broad terminal S wave (due to delayed right ventricular activation), which may give the appearance of a negative QRS complex in lead I. Hence the S wave is not considered when axis is determined, as the axis reflects left ventricular activation. Nevertheless, in this case even when the S wave is not considered, the QRS complex is still negative in lead I. There is a suggestion of a P wave noted in leads III and aVL (▼), with the appearance of a waveform right after the QRS complex. If this is a P wave, the PR interval is 0.28 (↔), which is longer than the sinus PR interval (first QRS complex), and the RP interval is 0.10 second. Hence this would be termed a short-RP tachycardia. The etiologies for a short-RP tachycardia include atrial tachycardia, atrial flutter with 2:1 AV block, an unusual form of a typical atrioventricular nodal tachycardia termed slow-slow (with a fast pathway that conducts retrogradely relatively slowly), sinus tachycardia with first-degree AV block, ectopic junctional tachycardia, and an atrioventricular reentrant tachycardia.

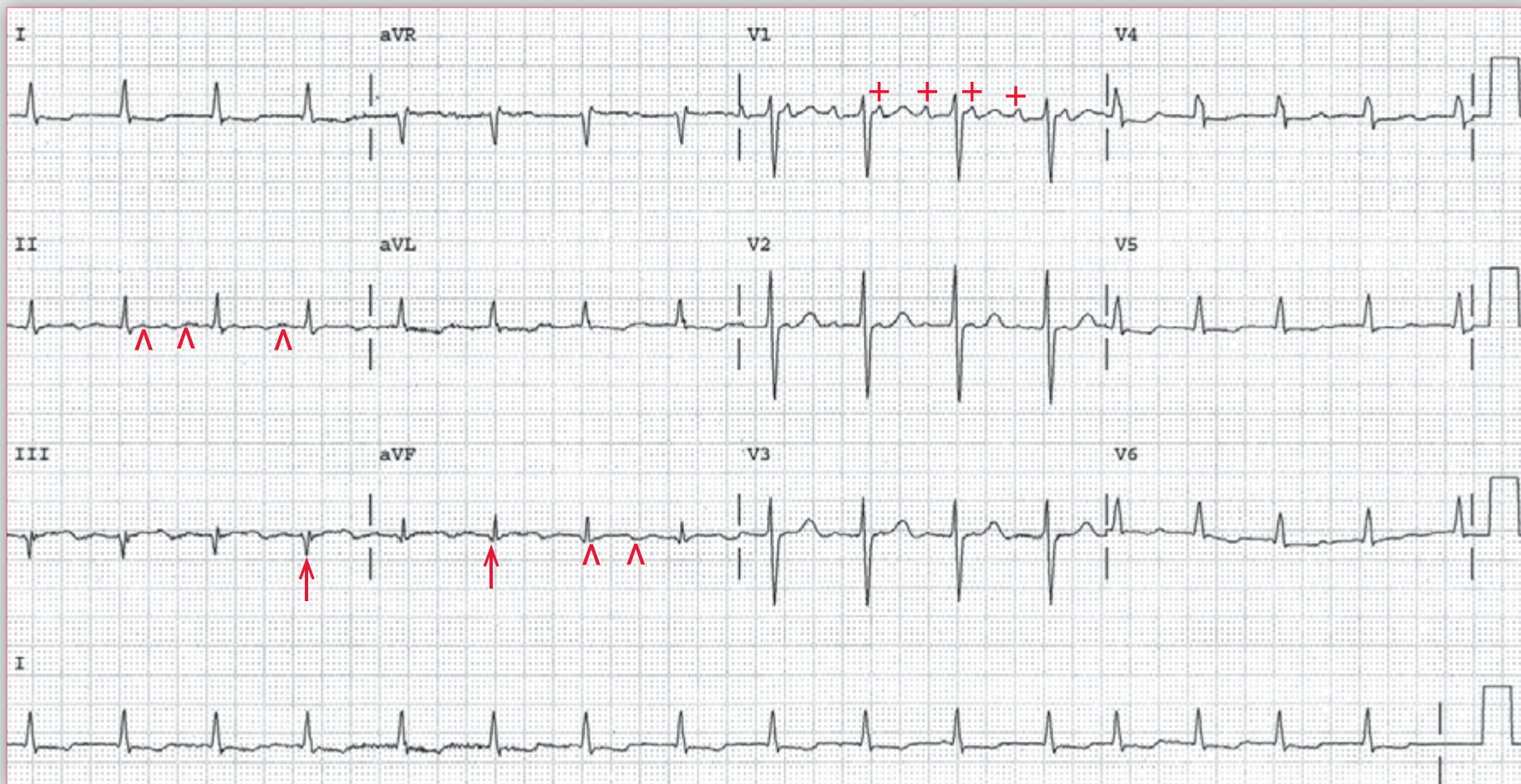
There is an abrupt slowing of the rate, and after the pause (⊐), the QRS complex duration is normal (0.08 sec) and there is no right bundle branch block pattern (↑). A P wave is seen before this narrow QRS

complex, best appreciated in lead V1 (^). The PR interval is 0.16 second. The QRS complex morphology and the PR interval are identical to that of the first QRS complex. Hence this is also a sinus QRS complex. After this sinus QRS complex, there is another P wave (seen in lead V1 (▲)), and it has a different morphology and is followed by a longer PR interval (0.28 sec) (⊐), which is identical to the PR interval measured in leads I, III, and aVF during the tachycardia (↔). Following this P wave, there is another episode of the tachycardia with a QRS complex that has a right bundle branch block morphology. The rate and QRS complex width and morphology are identical to those during the first episode of tachycardia. Although there are no obvious P waves seen, if the PR interval in lead V1 at the onset of the tachycardia is used (*ie*, 0.28 sec) (⊐), it can be seen that the rounded waveform at the end of the QRS complex in lead V1 (↓) is the P wave with a PR interval of 0.28 second (↔). This can be confirmed by measuring the QRS duration in lead V2 and comparing this to the QRS in lead V1 (||); the waveform at the end of the QRS complex is not part of the QRS and is indeed the P wave. This is an atrial tachycardia with 1:1 conduction and a rate-related right bundle branch block.

There appear to be Q waves in leads II, III, and aVF (^), suggesting an old inferior wall myocardial infarction. There is low voltage, defined as a QRS complex amplitude that is < 5 mm in each limb lead and < 10 mm in each precordial lead.

continues

Podrid's Real-World ECGs



ECG 10B Analysis: Atrial tachycardia with 2:1 AV block, possible old inferior wall infarction

ECG 10B is from the same patient as ECG 10A. There is a regular rhythm at a rate of 94 bpm. The QRS complex duration is normal (0.08 sec) and there is a normal morphology. The morphology is the same as that of the narrow QRS complexes in ECG 10A. The QT/QTc intervals are normal (340/430 msec). There are distinct P waves seen in lead V1–V2 (+) and the atrial rate is 180 bpm, which is identical to the ventricular rate seen in ECG 10A. There is 2:1 AV block. The P waves appear to be negative–positive in leads II and aVF (^). Hence this is an atrial tachycardia with 2:1 AV block, confirming the fact that ECG 10A showed an atrial tachycardia with 1:1 conduction. The QRS complex duration is normal (0.08 sec), the axis is normal between 0° and +90° and the QRS complexes do not have a right bundle branch block morphology. This confirms the fact that ECG 10A showed an atrial tachycardia at a rate-related right bundle branch block. There

are small Q waves in leads III and aVF (↑), again suggesting an old inferior wall myocardial infarction. The QT/QTc intervals are normal (340/425 msec).

Carotid sinus pressure, as well as a Valsalva maneuver, transiently enhances vagal tone to slow conduction through the AV node. As a result there may be transient AV nodal block that allows identification of atrial activity. Once the atrial rate and P-wave morphology is observed, the etiology of the arrhythmia can be established. A similar response will occur with adenosine or any other AV nodal blocking agent such as a β -blocker, calcium-channel blocker (verapamil or diltiazem), or digoxin, although with these agents the AV nodal blockade will be longer lasting. Importantly, none of these maneuvers or drugs will terminate the atrial tachycardia or slow the atrial rate. ■

Core Case 11

A 64-year-old woman presents to her local emergency department with acute onset of chest pain. She notes sudden onset of mid-sternal “heaviness” associated with dyspnea one hour before arriving at the emergency department. She dialed 911 for assistance.

ECG 11A



Upon arrival in the emergency department, she appears pale and in obvious discomfort but conscious and able to respond to questioning. Her vital signs reveal a marked tachycardia and low blood pressure. As part of her initial workup, an ECG is obtained (ECG 11A). She is treated with intravenous medications and her symptoms abruptly resolve. A follow-up ECG is obtained (ECG 11B).

What etiologies for the patient's symptoms are suggested?

What diagnosis can now be confirmed?

ECG 11B



Podrid's Real-World ECGs



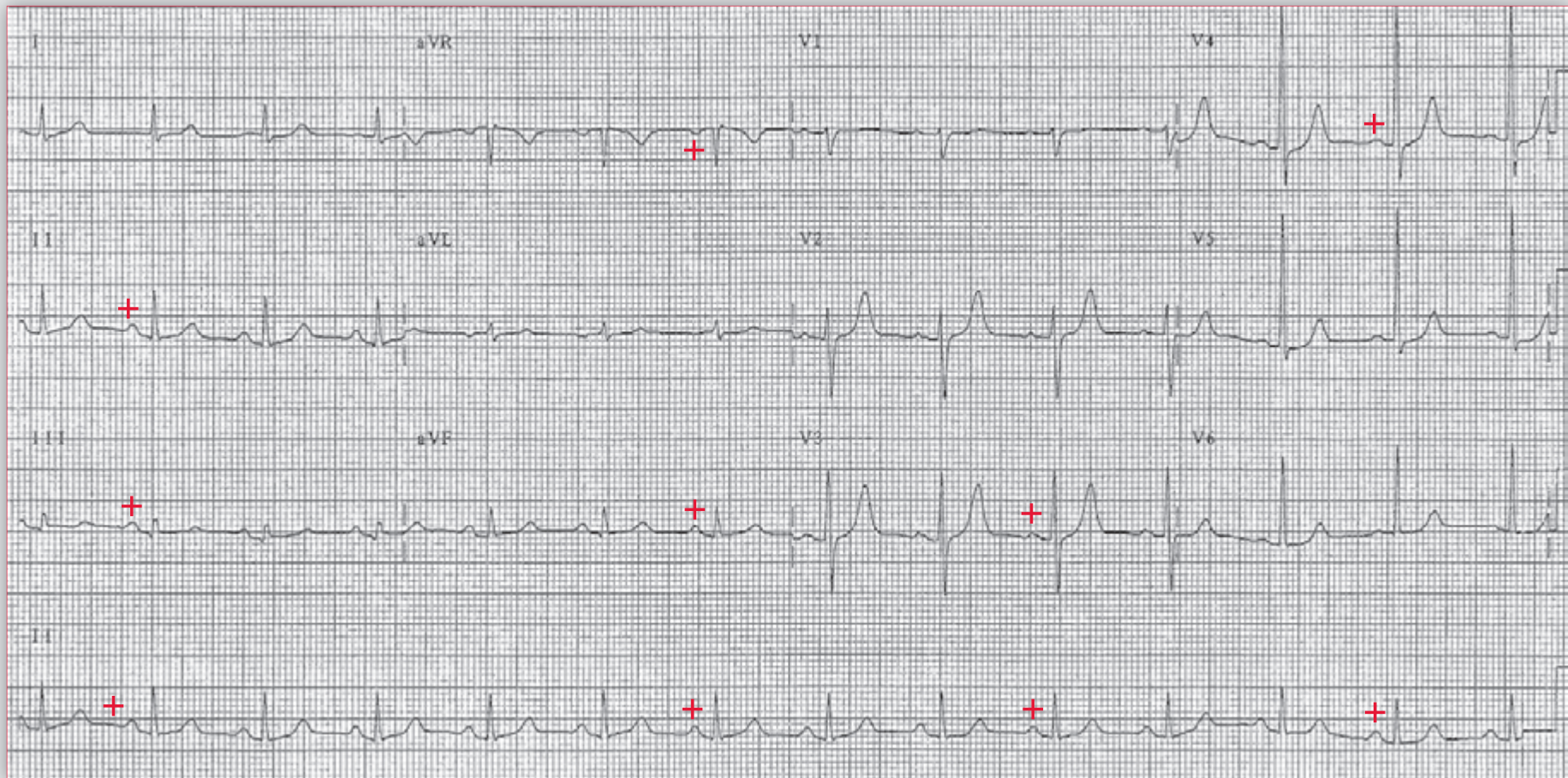
ECG 11A Analysis: Wide complex tachycardia due to atrial flutter with 1:1 conduction, right bundle branch, left posterior fascicular block

ECG 11A shows a regular rhythm at a rate of 270 bpm. There are no obvious P waves seen. The QRS morphology has a typical right bundle branch block morphology (RSR' in V1 [→] and broad S waves in leads I and V4–V6 [←]). The axis is rightward, between +90° and +180° (negative QRS complex in lead I and positive QRS complex in lead aVF). In the presence of a right bundle branch block, there is a broad terminal S wave (due to delayed right ventricular activation), which may give the appearance of a negative QRS complex in lead I. Hence the S wave is not considered when axis is determined as the axis reflects left ventricular activation. Nevertheless, in this case even when the S wave is not considered, the QRS complex is still negative in lead I. The only two rhythms that present with this rate are atrial flutter (with 1:1 AV

conduction) or ventricular tachycardia, which is often termed ventricular flutter when it occurs at this rate. The uniformity of the QRS complexes, the presence of a typical right bundle branch block, and the fact that in leads V4–V5 the R wave is narrower than the S wave (*ie*, $R/S < 1$) makes this a supraventricular rhythm, and hence atrial flutter with 1:1 conduction. Another finding is the presence of electrical or QRS complex alternans, particularly evident in leads V3–V4. Electrical alternans, which is often associated with a large pericardial effusion or tamponade, may also be seen in other situations, including an acute myocardial infarction, dilated cardiomyopathy, congestive heart failure or any rapid supraventricular tachyarrhythmia.

continues

Podrid's Real-World ECGs



ECG 11B Analysis: Normal sinus rhythm, normal ECG

ECG 11B is from the same patient as ECG 11A. There is a regular rhythm at a rate of 84 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.20 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is normal (0.08 sec) and there is a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QRS morphology is normal. The QT/QTc intervals are normal (340/400 msec). The presence of a normal QRS complex at a slower rate confirms the fact that ECG 11A showed atrial flutter with a rate-related right bundle branch block.

Atrial flutter is the result of a reentrant circuit within the right atrium. The atrial rate is 260 to 320 bpm. Hence any supraventricular rhythm at a regular atrial rate within this range is atrial flutter. When the ventricular rate is also in this range with a QRS complex that is supraventricular, the diagnosis is atrial flutter with 1:1 conduction.

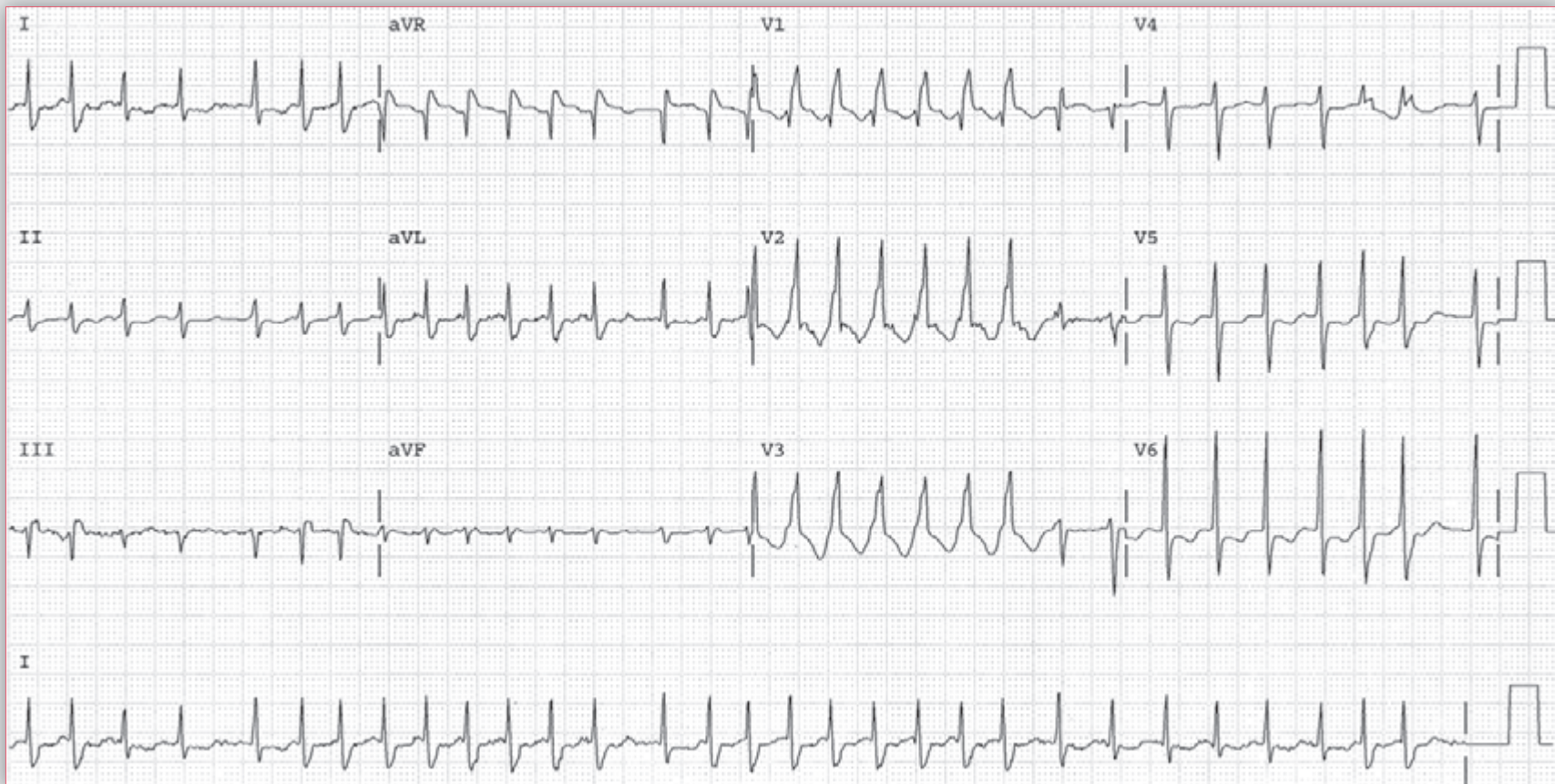
Although not very common, 1:1 conduction results from any situation in which there is enhanced AV nodal conduction, and this is primarily when there is sympathetic nervous system activation or an increase in circulating catecholamine levels. As the atrial flutter may be the result of sympathetic activation, there is a possibility that a β -blocker may not only slow the ventricular response rate, but may also result in termination of the arrhythmia. The treatment of atrial flutter is first to slow the ventricular rate with an AV nodal blocking agent such as a β -blocker, calcium-channel blocker (verapamil or diltiazem), or digoxin. Adenosine has only a transient effect and hence is useful for diagnosis but not treatment. The arrhythmia can be reverted with electrocardioversion or with an antiarrhythmic drug that is class IA (IV procainamide, quinidine or disopyramide), class IC (propafenone or flecainide), or a class III (IV ibutilide, IV amiodarone, sotalol, or dofetilide). ■

Notes

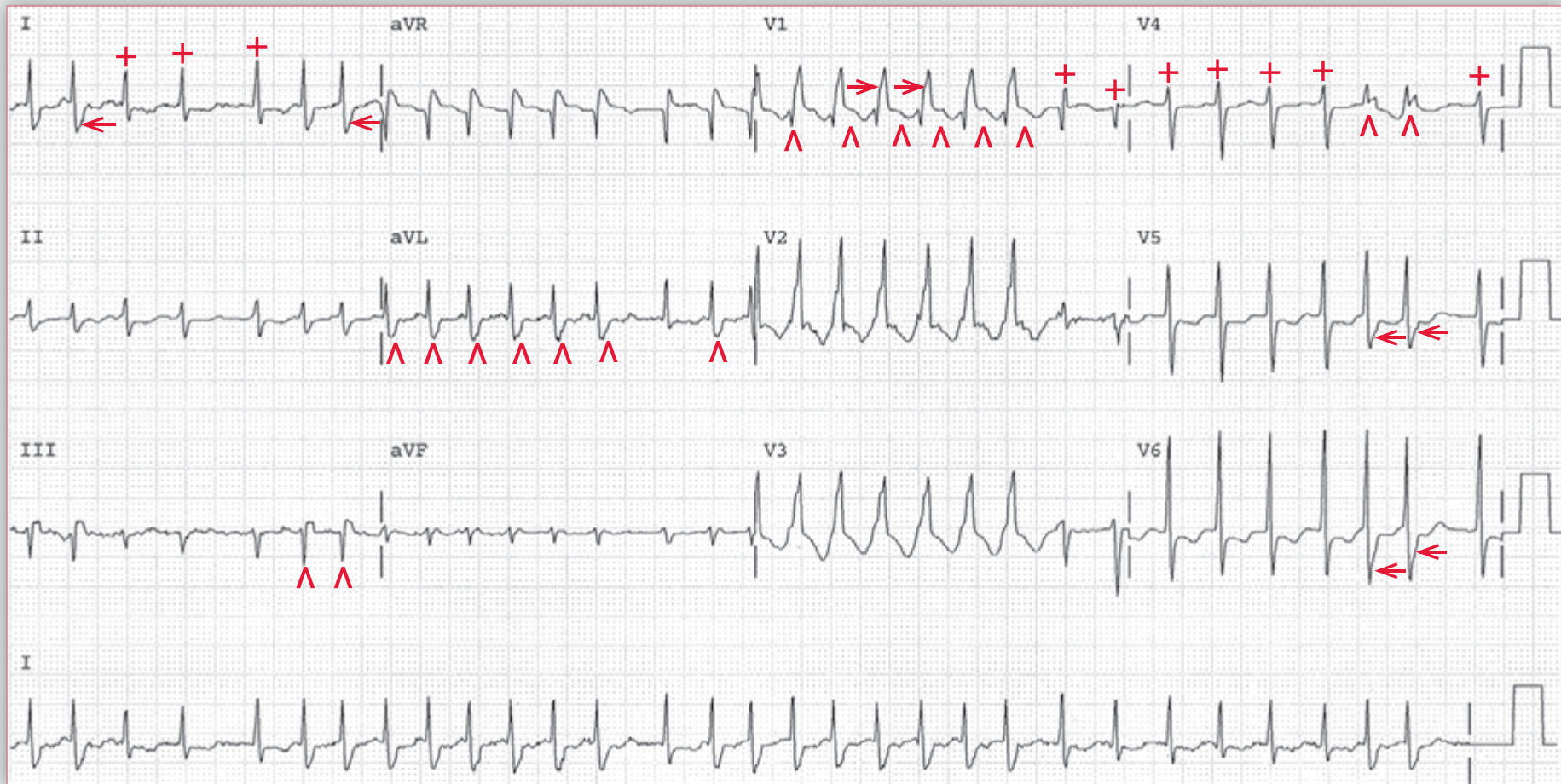
A 56-year-old woman presents to her primary doctor as an urgent care visit. She has had palpitations and chest pressure for several hours since waking. Her radial pulse is rapid and her JVP is elevated. Her lungs are clear. An ECG is obtained.

What abnormalities are noted on her tracing?

What initial therapy should be provided?



Podrid's Real-World ECGs



ECG 12 Analysis: Atrial fibrillation with rapid ventricular response, rate-related right bundle branch block

The rhythm is irregularly irregular at a rate of 186 bpm. There are only three supraventricular rhythms that are irregularly irregular: sinus arrhythmia, in which there is one P-wave morphology and a stable PR interval; multifocal atrial tachycardia (rate > 100 bpm) or multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm), in which there are three or more P-wave morphologies without any dominant P wave; or atrial fibrillation, in which there are no organized P waves seen. In this ECG, there are no P waves seen before or after any QRS complex. Hence this is atrial fibrillation with a rapid ventricular response. The QRS complexes have two different widths and morphologies. The narrow complexes (+) have a duration of 0.08 second and have a normal morphology. The wide QRS complexes (^) have a duration of 0.12 second and these QRS complexes have a right bundle branch block morphology (RSR' in V1 [→] and broad S wave in leads I and V5–V6 [←]). The axis is physiologically leftward between 0° and –30° (positive QRS complex in leads I and II and negative in lead aVF).

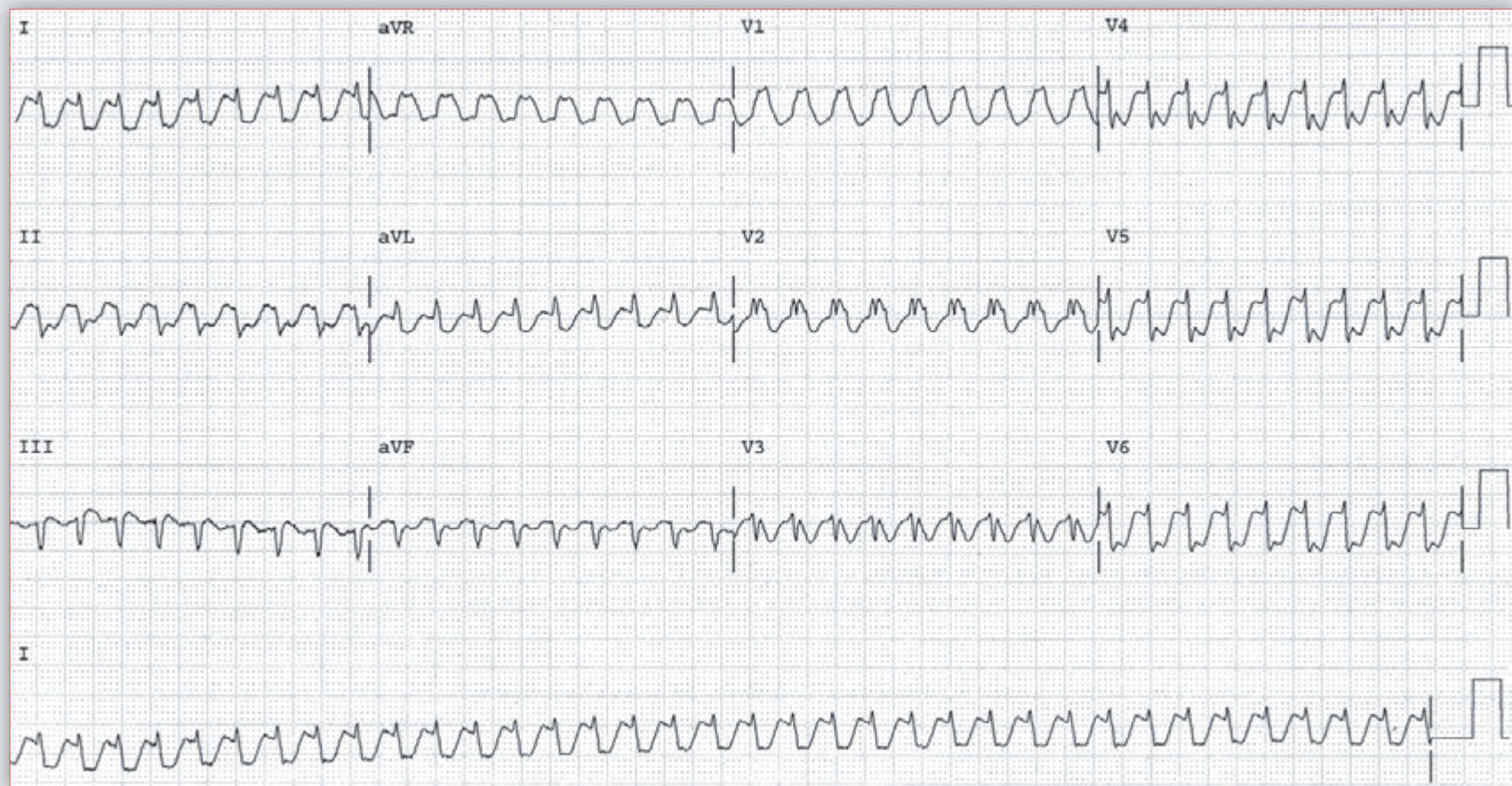
It can be seen that the wide QRS complexes with a right bundle branch block pattern are associated with shorter RR intervals (or faster rate). Hence this is a rate-related right bundle branch block.

As the symptoms with atrial fibrillation are usually due to the rapid ventricular response rate, initial therapy is slowing the ventricular rate. As the ventricular rate is due solely to AV nodal conduction, initial therapy is directed at AV nodal blockade to slow conduction through this structure and thereby slow the ventricular rate. This can be achieved with an AV nodal blocking agent, including a β -blocker, calcium-channel blocker (verapamil or diltiazem), or digoxin. Adenosine blocks the AV node, but its effect is very brief and hence this is not a therapeutic agent in this arrhythmia. ■

Core Case 13

A 35-year-old man presents to his community emergency department with complaints of a racing heart. Symptoms began that evening while he was watching television and have persisted since. He is otherwise healthy, on a swim team, and denies any functional limitation. He does not take any medications. His family history is unremarkable.

ECG 13A



He appears nervous but in no acute distress. His physical exam is unremarkable save for pectus excavatum and marked tachycardia on cardiac auscultation.

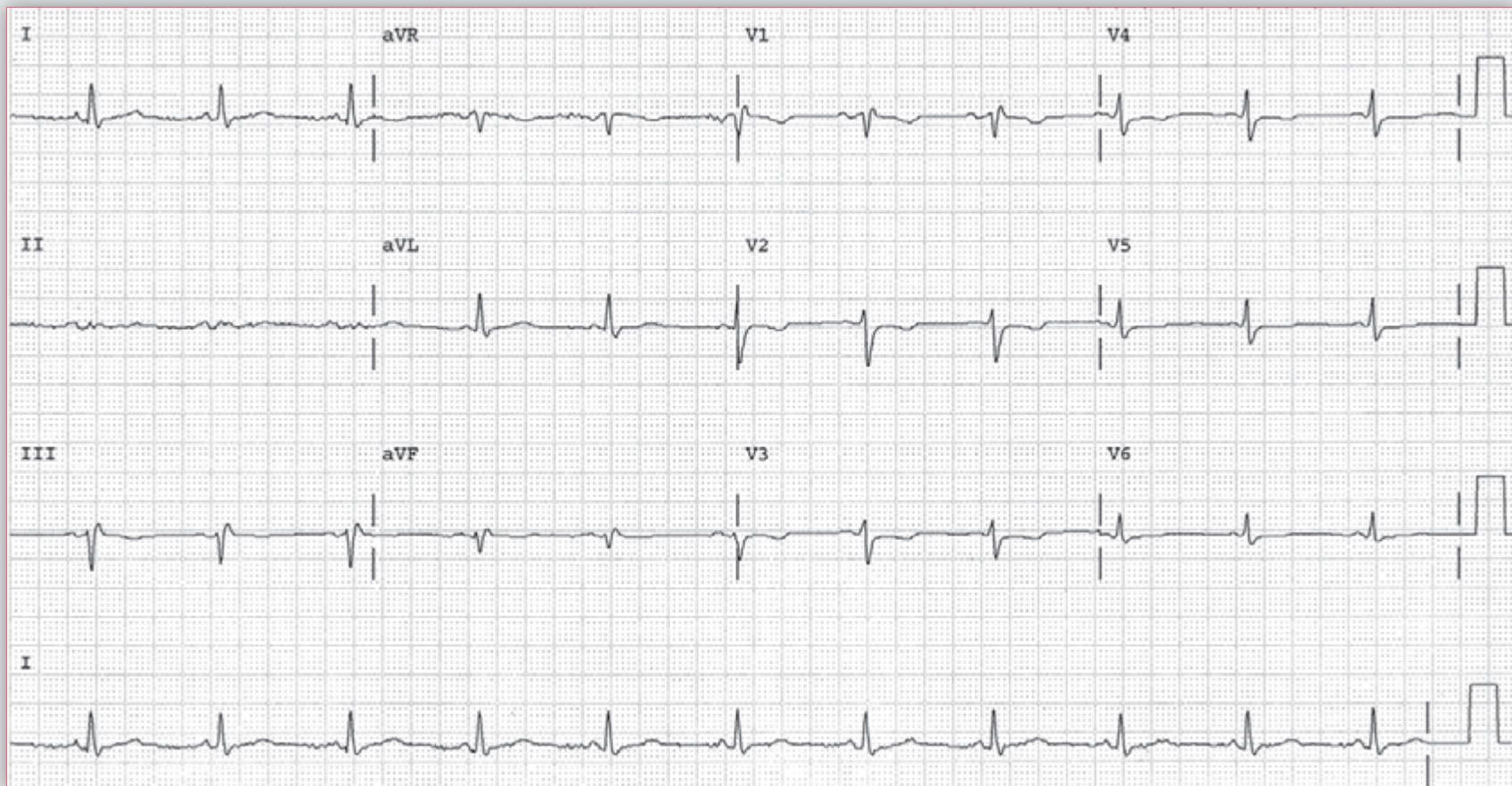
An ECG is obtained (ECG 13A). He is treated with medications and a follow-up ECG is obtained upon cessation of symptoms (ECG 13B).

What is the differential diagnosis?

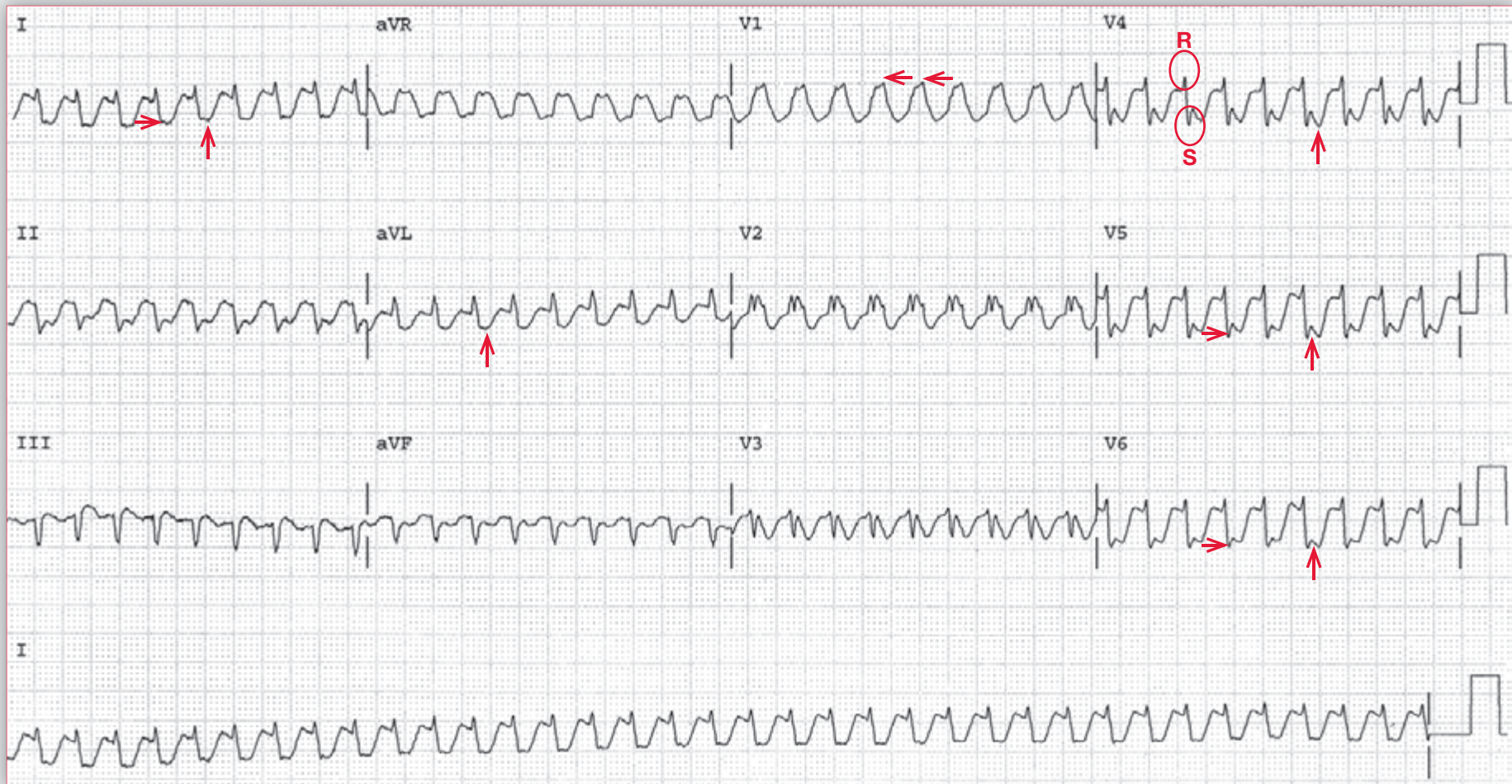
What diagnosis is now suggested when comparing tracings (ECG 13A and ECG 13B) in the context of the history?

What medication is likely to have been given?

ECG 13B



Podrid's Real-World ECGs



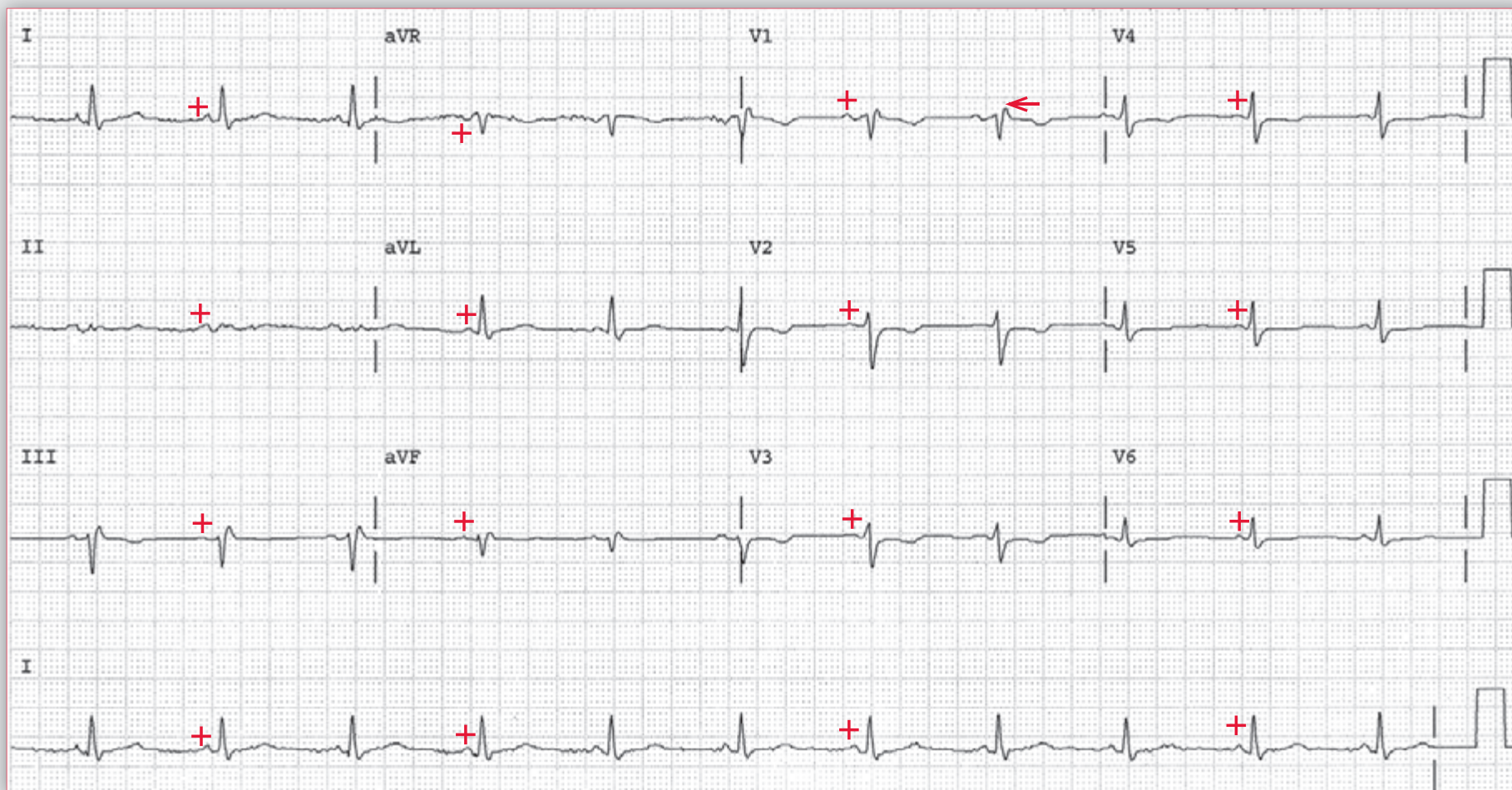
ECG 13A Analysis: Wide complex tachycardia due to an atrioventricular nodal reentrant tachycardia, right bundle branch morphology, left anterior fascicular block, ST-segment depressions, low voltage

ECG 13A shows a regular rhythm at a rate of 220 bpm. There are no P waves seen before or after any QRS complex. The QRS complex duration is increased (0.16 sec) and there is a right bundle branch block morphology (broad R wave in lead V1 [←] and broad S waves in leads I and V4–V6 [→]). Noted is that the initial R wave in leads V4–V6 is narrow (< 100 msec) while the S wave is widened and accounts for the increase in the QRS duration. Hence this is a criterion for a supraventricular tachycardia with a right bundle branch block. In addition, the axis is extremely leftward between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF). The two etiologies for an extreme left axis are an inferior wall myocardial infarction, in which there is a deep initial Q wave in leads II and aVF, or a left anterior fascicular block, in which there is an rS QRS morphology in leads II and aVF. Therefore, this is a left anterior fascicular block. There is possible ST-segment depression seen in leads I, aVL, and V4–V6 (↑), although it is not clear where the S wave ends and the ST segment begins. Noted is low voltage, defined as a QRS complex amplitude < 5 mm (little boxes) in each limb lead and/or < 10 mm in each precordial lead. With the absence of P waves, this is termed a no-RP tachycardia.

The absence of atrial activity and the rate of 220 bpm of the supraventricular rhythm makes the most likely etiology a typical atrioventricular reentrant tachycardia (AVNRT). This arrhythmia is due to the presence of dual AV nodal pathways. There is a fast pathway, which conducts rapidly but has a long refractory period and hence recovers slowly. The other pathway is a slow pathway that conducts slowly, but recovers more quickly. With a typical AVNRT the antegrade conduction to the ventricles is via the slow pathway while the retrograde conduction back to the atria is via the fast pathway. Hence the typical AVNRT is termed slow-fast. There is simultaneous activation of the atria and ventricles, resulting in the absence of obvious P waves (no-RP tachycardia) as they are occurring at the same time as ventricular activation, *ie*, simultaneously with the QRS complex. AVNRT is usually initiated by a premature atrial complex. If this premature atrial complex occurs early enough, such that the fast pathway has not yet recovered, it is blocked in the fast pathway, but conducts to the ventricles via the slow pathway; thus there is a very long PR interval. If by the time the impulse reaches the terminal portion of the circuit, the fast pathway has recovered, the impulse enters the fast pathway retrogradely, activating the atrium at the same time it is conducted antegradely to the ventricles via the His-Purkinje system. If the slow pathway has recovered by the time the impulse reaches the proximal portion of the circuit, the impulse can reenter the slow pathway. This accounts for the terms slow-fast.

continues

Podrid's Real-World ECGs



ECG 13B Analysis: Normal sinus rhythm, normal ECG, low-voltage precordial leads, left axis, slight conduction delay to the right ventricle, low voltage.

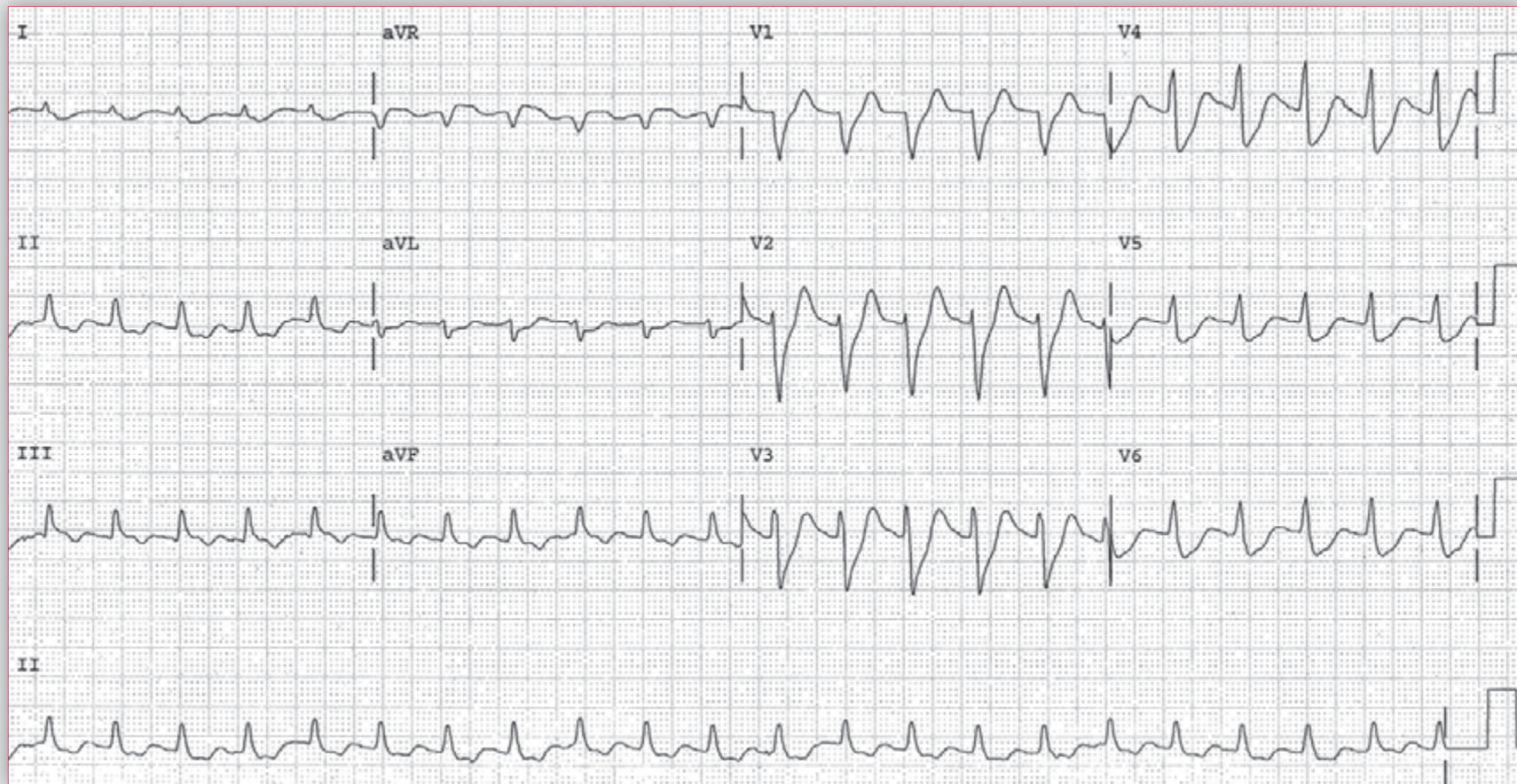
ECG 13B is from the same patient as ECG 13A. There is a regular rhythm at a rate of 66 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, aVF, and V4–V6; hence this is a normal sinus rhythm. The QRS complex duration is normal (0.10 sec) and there is an R' in lead V1 (←), which is the result of an intraventricular conduction delay to the right ventricle. The axis is leftward between 0° and –30° (positive QRS complex in leads I and II and negative in lead aVF). The QT/QTc intervals are normal (410/430 msec). There is diffuse low-voltage (QRS complex < 5 mm or little boxes in each limb lead and < 10 mm in each precordial lead). The right bundle branch block morphology is no longer present as the rate is slow. This confirms the fact that ECG 13A showed an AVNRT with a rate-related right bundle branch block aberration.

An AVNRT can be terminated whenever there are changes of conduction of either the slow or the fast pathway, although the slow pathway is usually the limb that is affected. Changes in the electrophysiologic properties can be produced by changing autonomic tone, for example, a vagal maneuver such as Valsalva or carotid sinus pressure will slow or block impulse conduction. Adenosine can also slow or block AV nodal conduction is an effective therapy for terminating this arrhythmia. AV nodal blocking agents such as β -blockers, calcium-channel blockers (verapamil or diltiazem), or digoxin are also effective. ■

Core Case 14

A 16-year-old male presents to his local emergency department with sudden-onset palpitations and dyspnea. He was home playing online video games when he suddenly became anxious and felt his “heart popping out of (his) chest.” His mother activated emergency services.

ECG 14A



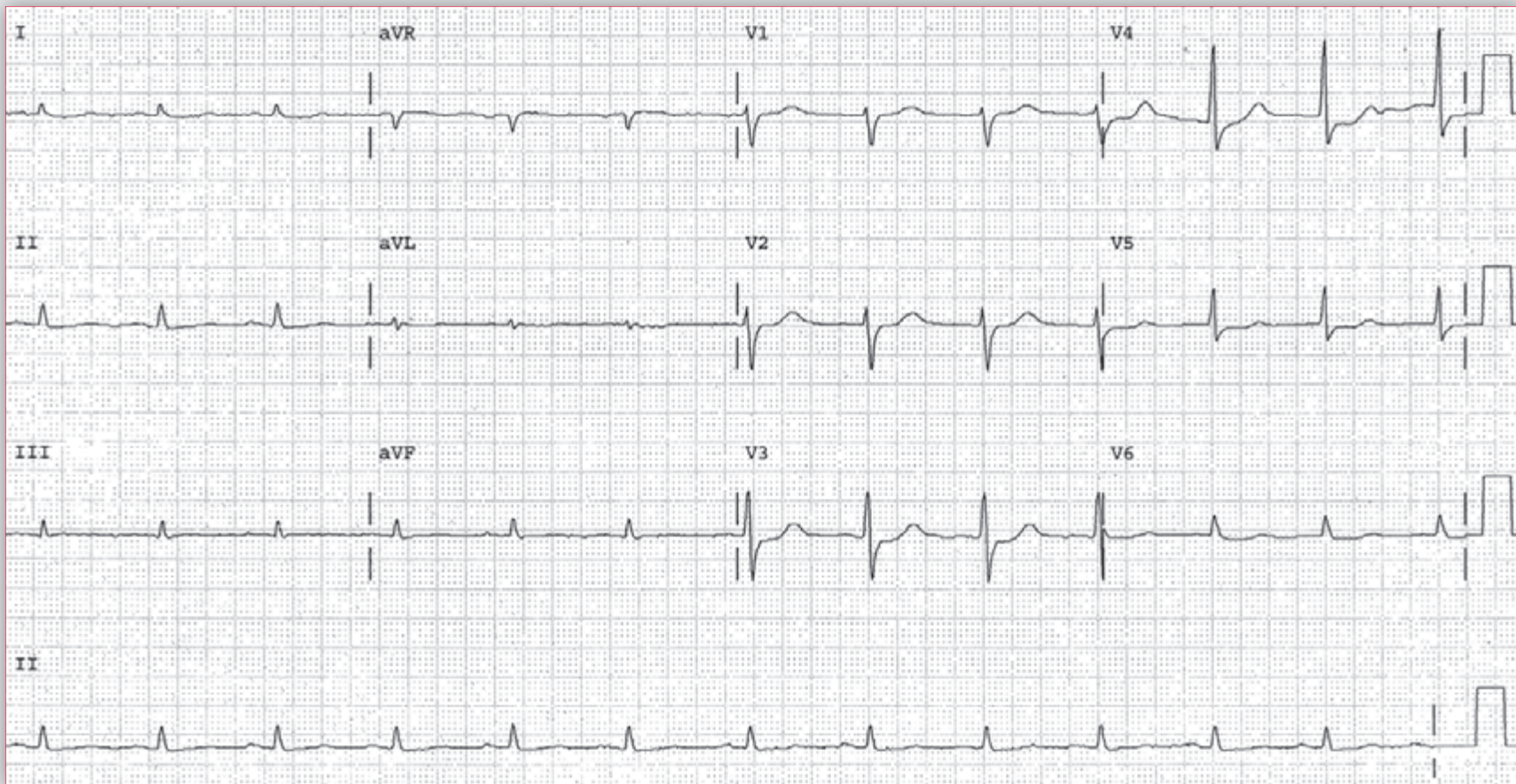
On presentation, he is an obese male in mild distress but otherwise normal in appearance. His exam is notable for acanthosis nigricans and rapid apical pulsations. As part of his workup, an ECG is obtained (ECG 14A).

He is treated with intravenous medication and his symptoms resolve. A follow-up tracing is obtained (ECG 14B).

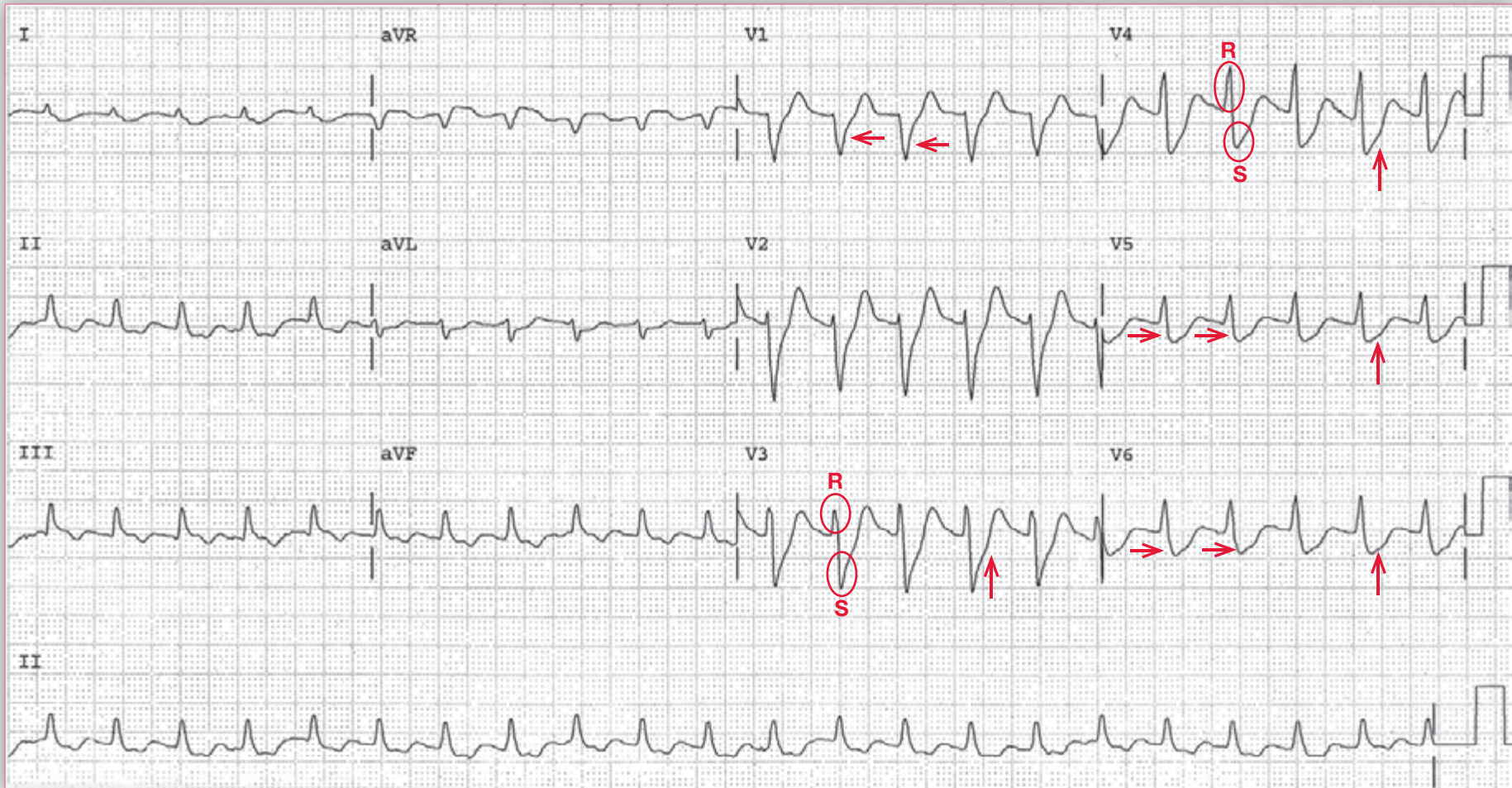
What is your interpretation of the tracing 14A and what electrical diagnosis is suggested?

What additional information does your interpretation of this tracing 14B provide?

ECG 14B



Podrid's Real-World ECGs



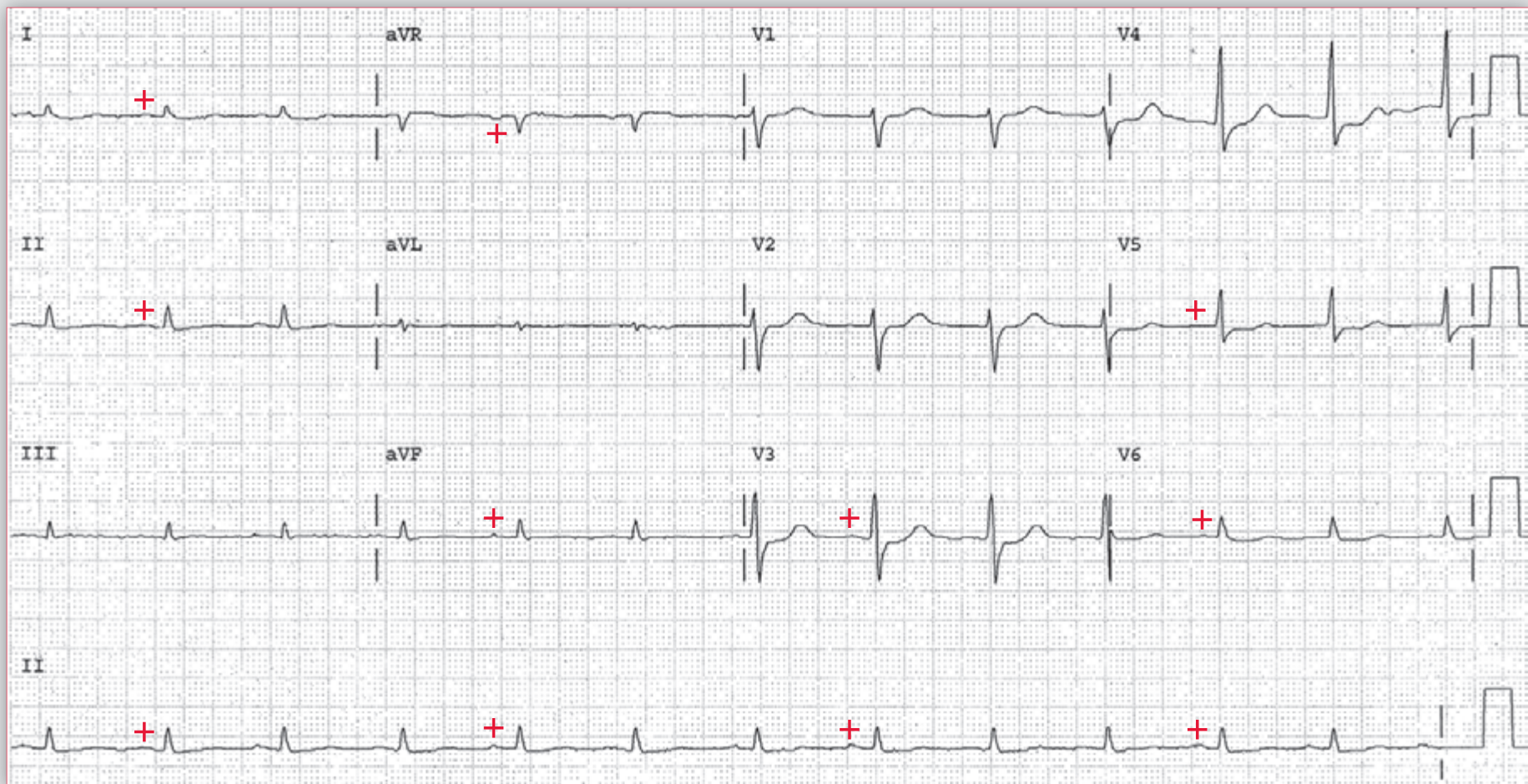
ECG 14A Analysis: Wide complex tachycardia due to atrioventricular nodal reentrant tachycardia, intraventricular conduction delay

ECG 14A shows a regular rhythm at a rate of 140 bpm. There are no P waves seen before or after any of the QRS complexes. The QRS complexes are wide, with a duration of 0.16 sec. They have a morphology that is not typical of either a left or right bundle branch block. While there is a QS complex in lead V1 (←), suggesting a left bundle branch block, there is a broad terminal S wave (→) in lead V5–V6, which is consistent with a right bundle branch block and terminal forces in a left-to-right direction. With a left bundle branch block all forces are directed from right to left and there are no left-to-right forces. Hence this is a nonspecific intraventricular conduction delay (IVCD). The axis is normal between 0° and +90° (positive QRS complex in leads I

and aVF). The QT/QTc intervals are prolonged (320/490 msec), but are normal when the widened QRS complex is considered (260/400 msec). It is not clear if this is a supraventricular QRS complex with aberration, or a ventricular complex. However, there are RS complexes noted in leads V3–V4. The R wave is narrower than the S wave (*ie*, R/S < 1) and in addition the R wave width is < 100 msec). This is consistent with a supraventricular tachycardia with aberration (IVCD). As P waves are absent, this is termed a no-RP tachycardia, and the most likely etiology is an atrioventricular nodal reentrant tachycardia (AVNRT). There are ST-segment depressions noted in leads V3–V6 (↑) which are related to the tachycardia and may be the result of subendocardial ischemia.

continues

Podrid's Real-World ECGs



ECG 14B Analysis: Normal sinus rhythm, low limb lead voltage

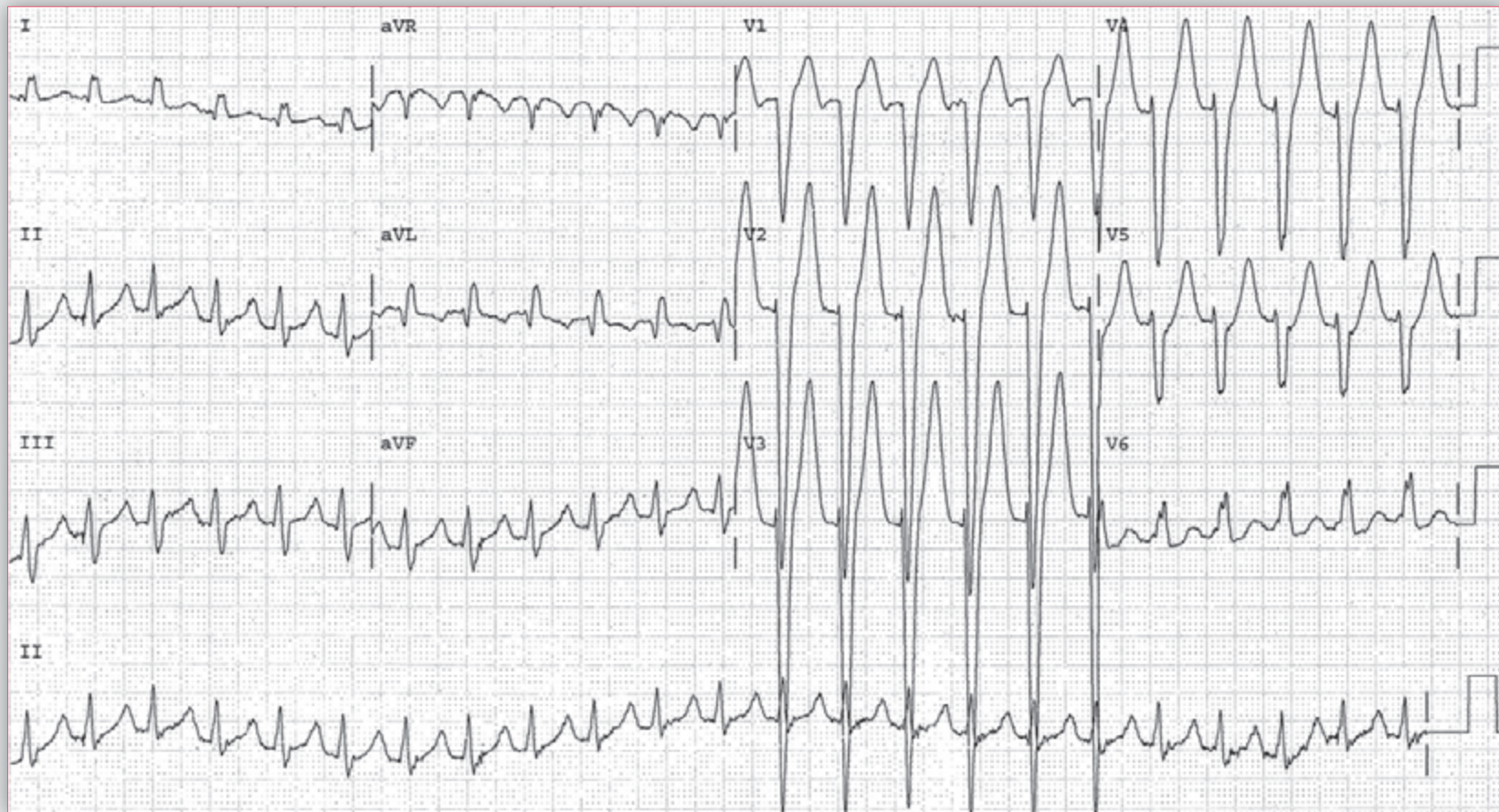
ECG 14B is from the same patient as ECG 14A. There is a regular rhythm at a rate of 76 bpm. P waves (+) can be seen before each QRS complex with a constant PR interval (0.18 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence, this is a normal sinus rhythm. The QRS complex duration is normal (0.10 sec), and there is a normal morphology. The axis is normal between 0° and +90° (QRS complex positive in leads I and aVF). The QT/QTc intervals are slightly prolonged (400/450 msec). There is low QRS complex voltage in the limb leads (< 5 mm in each lead). The fact that the QRS complex width and morphology is normal at this rate confirms that ECG 14 showed a rate-related IVCD.

An AVNRT can be terminated whenever there are changes of conduction of either the slow or the fast pathway, although the slow pathway is usually the limb that is affected. Changes in the electrophysiologic properties can be produced by changing autonomic tone, for example a vagal maneuver such as Valsalva or carotid sinus pressure will slow or block impulse conduction. Adenosine can also slow or block AV nodal conduction is an effective therapy for terminating this arrhythmia. AV nodal blocking agents such as β -blockers, calcium-channel blockers (verapamil or diltiazem), or digoxin are also effective. ■

Core Case 15

A 72-year-old man is admitted to hospital with pneumonia. His medical history is notable only for hypertension. He does not take any medications for this. In fact, this is his first hospitalization and he has not followed with a primary doctor for decades.

ECG 15A



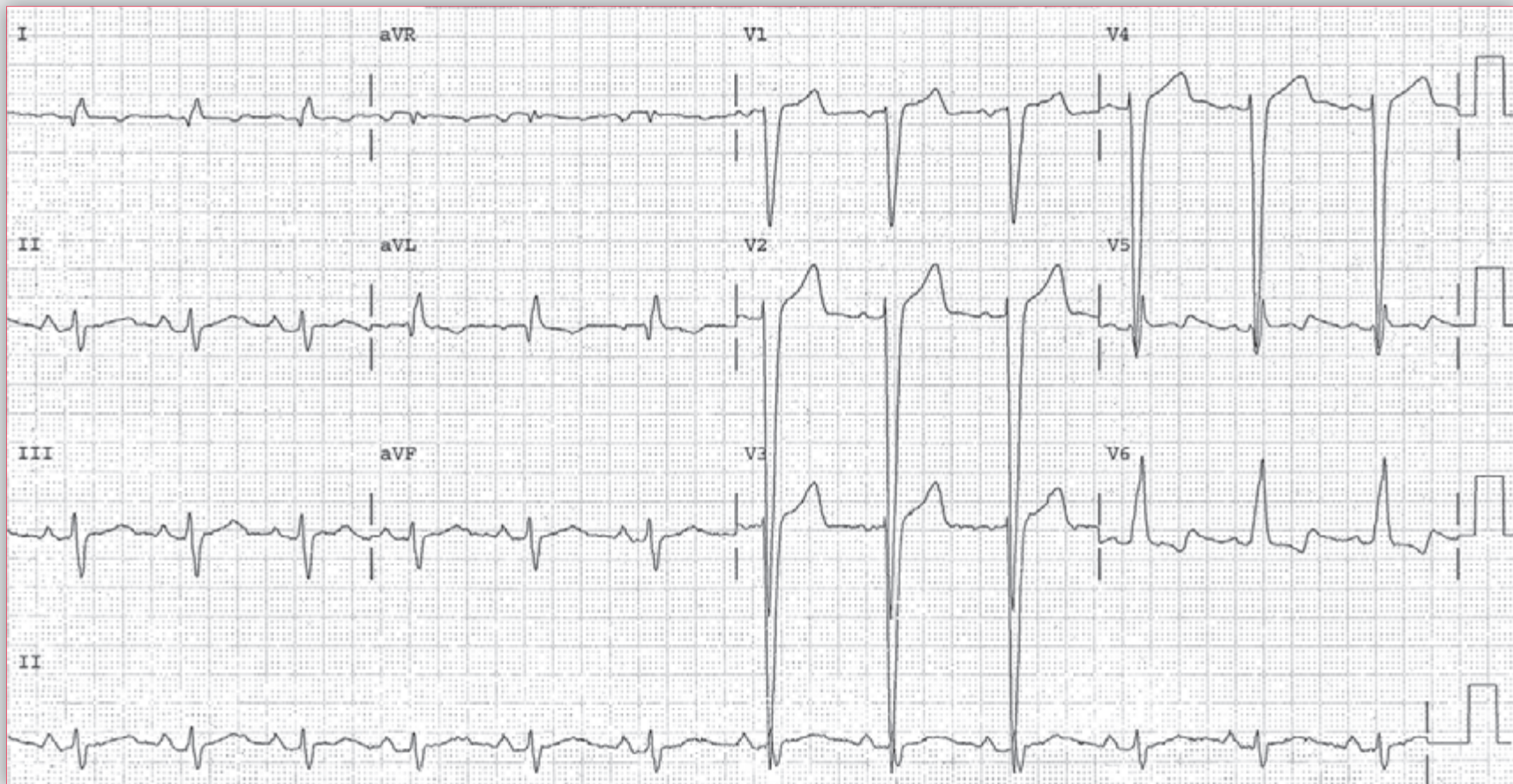
During his hospitalization, he becomes delirious. During one episode of nocturnal delirium, he complains of chest pain and an ECG is obtained (ECG 15A).

The following day upon the return of lucidity, a follow-up ECG is obtained (ECG 15B).

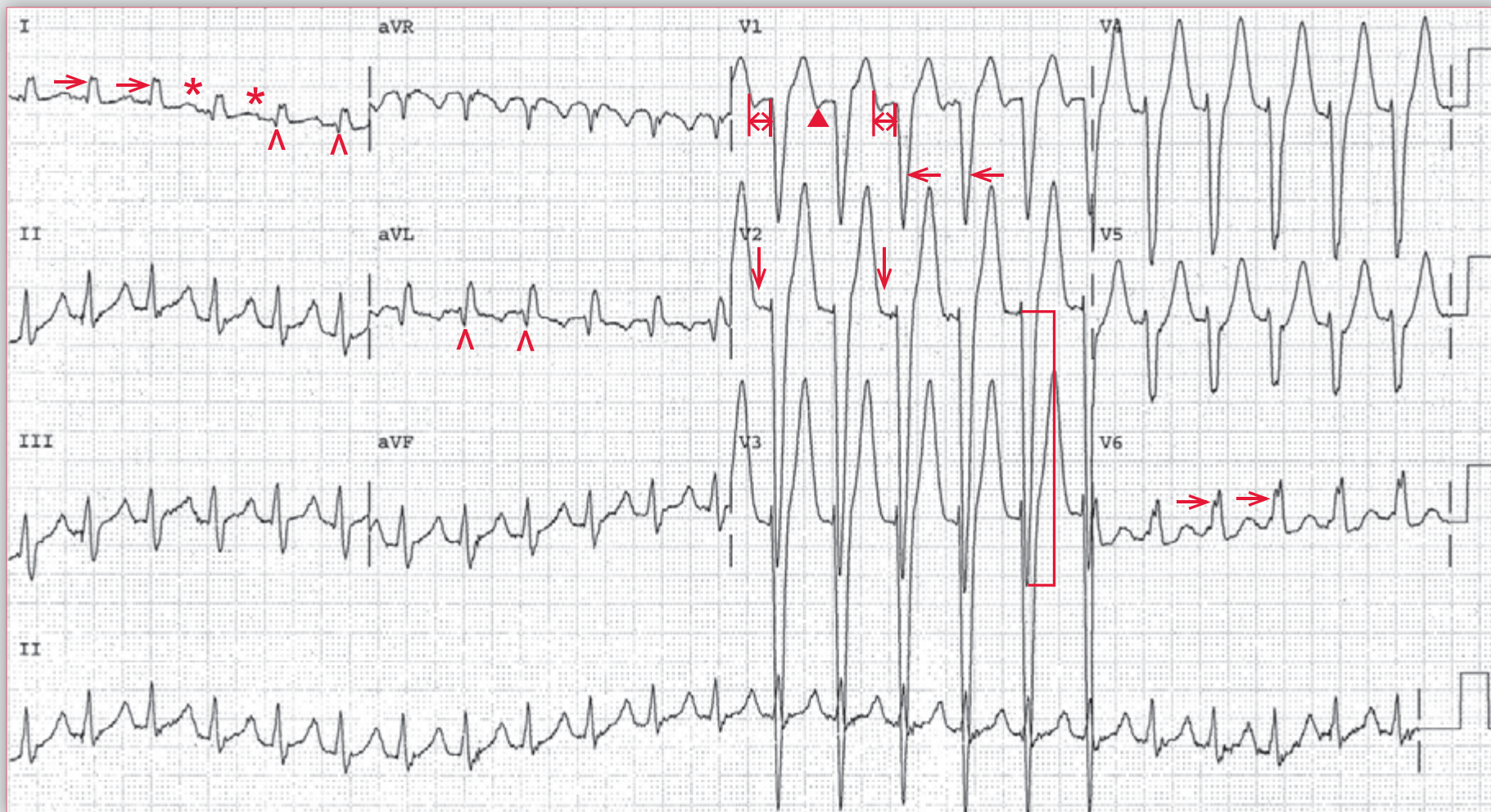
What is your interpretation of the tracing (ECG 15A)?

Correlating the two tracings (ECG 15A and ECG 15B), what diagnoses can be made?

ECG 15B



Podrid's Real-World ECGs



ECG 15A Analysis: Sinus tachycardia with intraventricular conduction delay, left ventricular hypertrophy

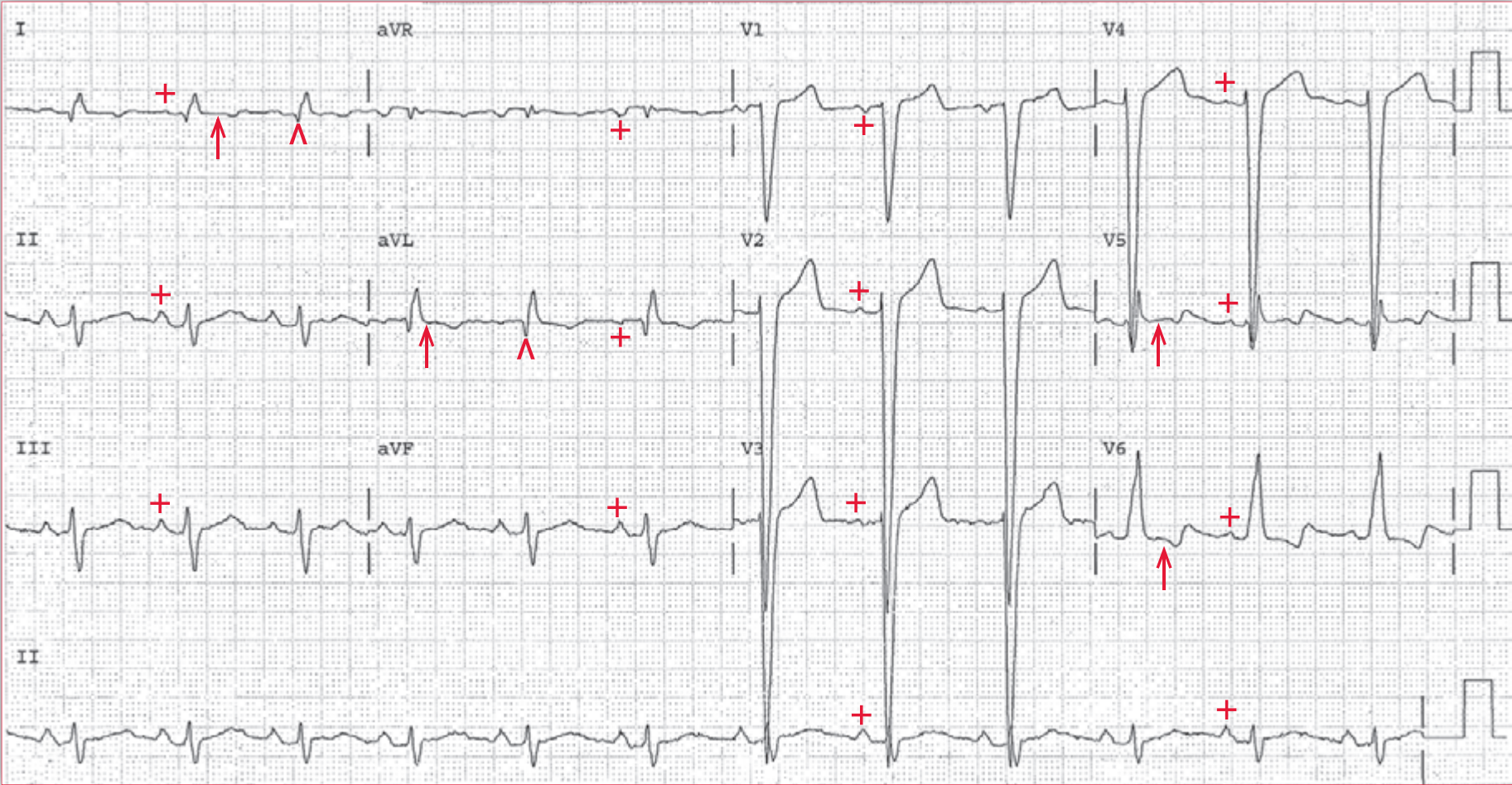
ECG 15A shows a regular rhythm at a rate of 140 bpm. There are no obvious P waves seen before or after any of the QRS complexes. However, there is a negative waveform seen after the T wave in lead V1 (▲). In addition, there is a subtle bump at the end of the T wave in lead V2 (↓), in lead V2 and notching of the T wave in lead I (+). This suggests a P wave superimposed at the end of the T wave. If this is a P wave, the PR interval (↔) is 0.18 second. The QRS complexes are wide, with a duration of 0.14 second. The QRS morphology resembles that of a left bundle branch block (broad R wave in leads I and V6 [→] and a QS complex in lead V1 [←]). However, there are septal Q waves seen in leads I and aVL (^). Septal Q waves cannot be seen with a left bundle branch block, as these forces are due to initial septal activation originating from a small septal branch that comes from the left bundle. Hence there is no initial septal activation with a left bundle branch block. Therefore, the QRS complex manifests a nonspecific intraventricular conduction delay (IVCD), which is the result of diffuse impulse slowing through the normal His-Purkinje system. The axis is normal

between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are prolonged (340/520 msec) but are only slightly prolonged when the prolonged QRS complex duration is considered (300/460 msec).

As the QRS complex has an IVCD, the activation of the ventricle uses the normal His-Purkinje system with slowed conduction. Since the activation is along the normal conduction system, abnormalities of the left ventricular myocardium can be identified. In contrast, with a left bundle branch block, the activation of the left ventricle is not along the normal conduction system, but occurs via an abnormal pathway directly utilizing the ventricular myocardium. Hence left ventricular abnormalities cannot be identified. As this case shows an IVCD, there is tall QRS complex voltage, *ie*, S wave in leads V2–V3 = 46 mm (]), consistent with left ventricular hypertrophy (*ie*, an R wave or S wave > 25 mm in any precordial lead).

continues

Podrid's Real-World ECGs



ECG 15B Analysis: Normal sinus rhythm, intraventricular conduction delay, left ventricular hypertrophy

ECG 15B is from the same patient as ECG 15A. There is a regular rhythm at a rate of 72 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). This is the same as the presumed PR interval in ECG 15A. Noted is the fact that there is no negative deflection seen after the T wave in lead V1, supporting the fact that this waveform seen in ECG 15A was indeed the P wave and not part of the T wave. The QRS complex morphology and width (0.14 sec) are identical to what was seen in ECG 15A. Although the QRS morphology resembles a left bundle branch block, there are septal Q waves in leads I and aVL (^); hence this is an intraventricular conduction delay (IVCD). The QRS amplitude in leads V2–V3 is the same as noted in ECG 15A; hence left ventricular hypertrophy is present. There are ST-T wave abnormalities noted in leads I, aVL, and V5–V6 (↑). These are likely secondary to left ventricular hypertrophy. The QT/QTc intervals are prolonged (460/500 msec) but are only slightly prolonged when the increased QRS duration is considered (420/460 msec).

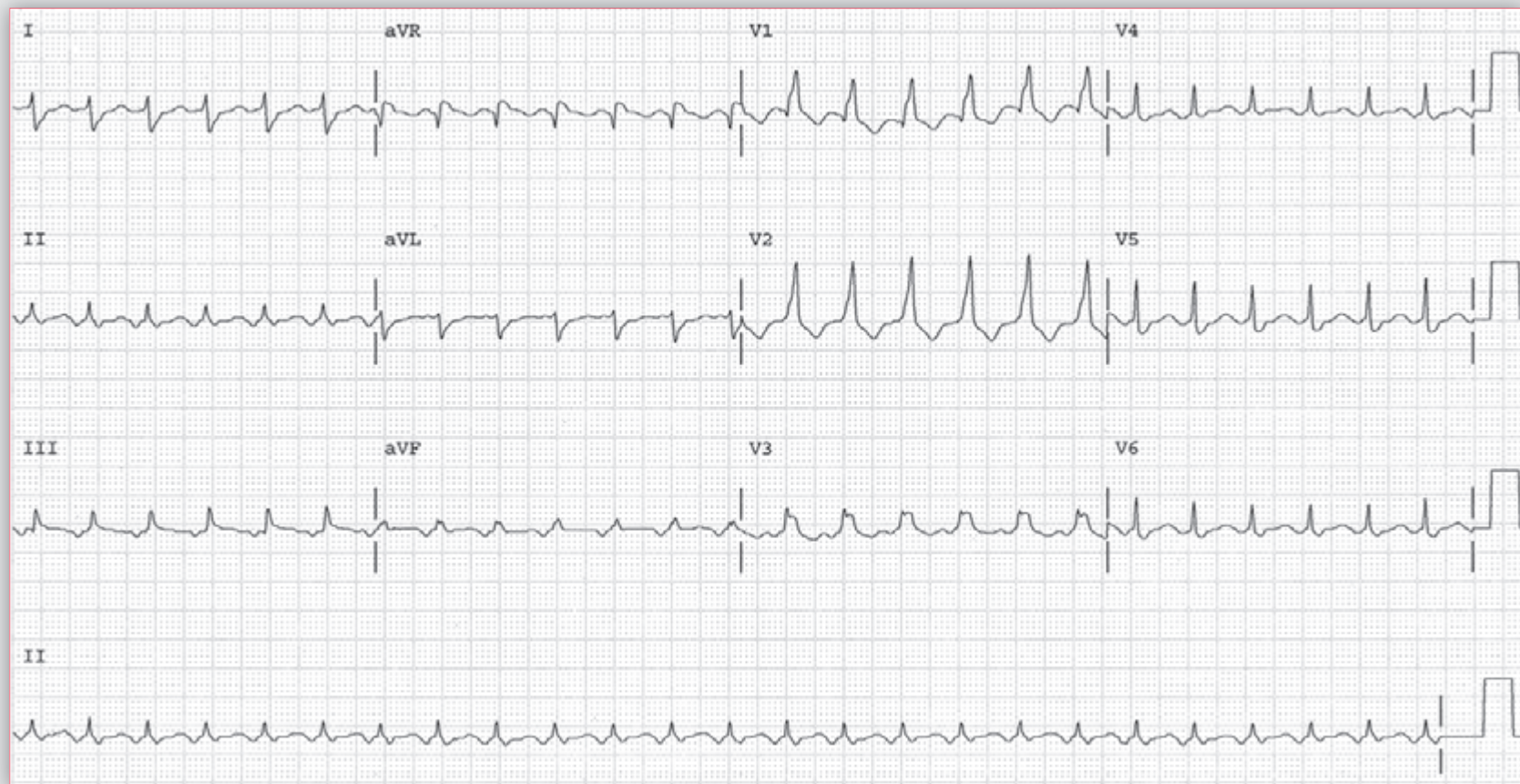
By comparing ECGs 15A with 15B, it can be seen that ECG 15A showed a sinus tachycardia with an IVCD that was also present at baseline during normal sinus rhythm. Hence this is not a rate-related aberration, but is an IVCD due to a preexisting IVCD.

Sinus tachycardia is always physiologic, the result of sympathetic activation or elevated catecholamines. It is likely that the sinus tachycardia is the result of the episode of delirium, which is generally associated with sympathetic activation. ■

Core Case 16

A 48-year-old woman without known medical history is admitted to hospital with abdominal pain. On the evening of her admission, she pages her nurse with complaints of sudden-onset palpitations. Her telemetry has also just alarmed. The nurse obtains a set of vital signs and an ECG while awaiting the responding clinician (ECG 16A).

ECG 16A

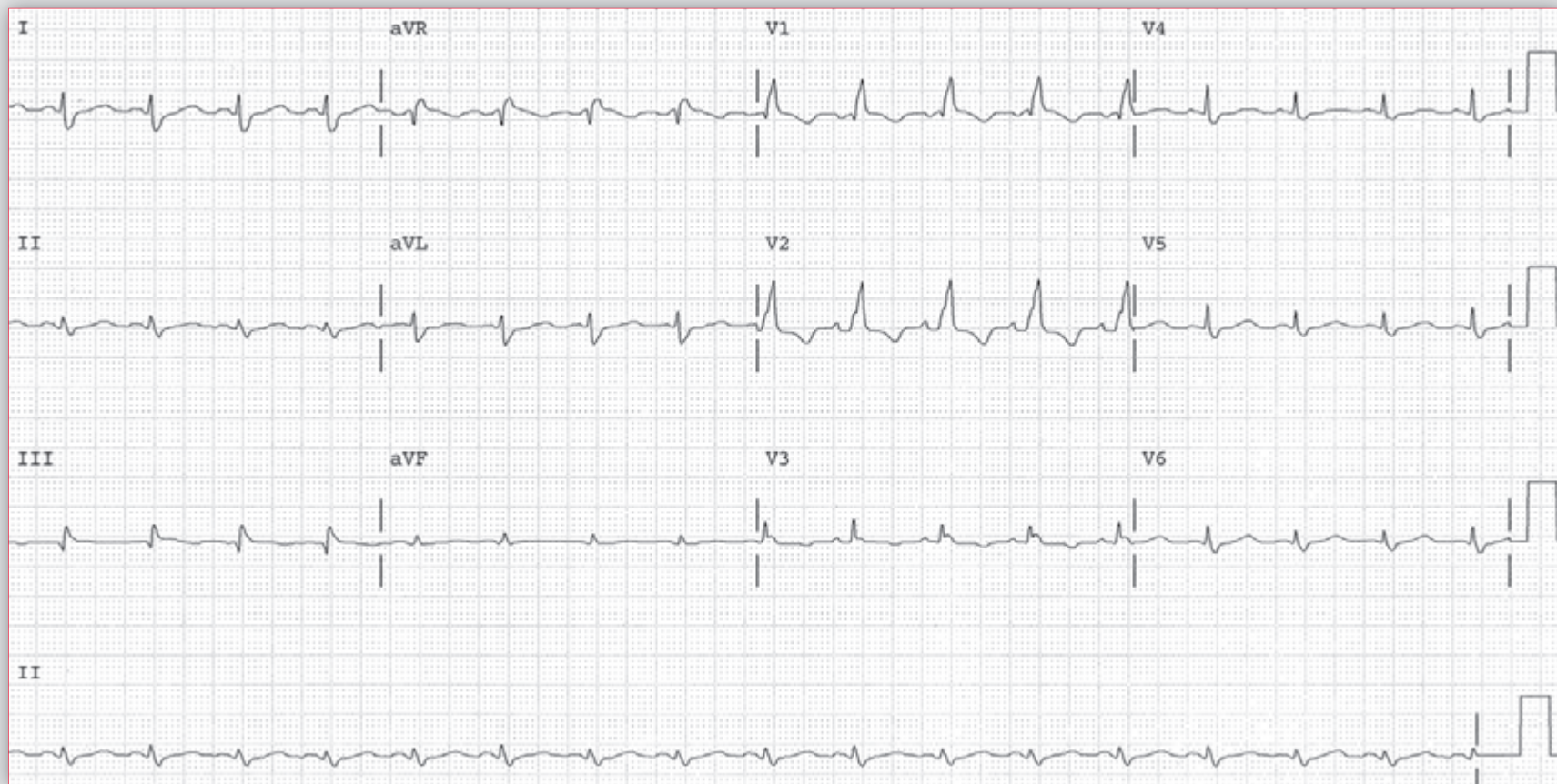


What is the differential diagnosis suggested by the ECG?

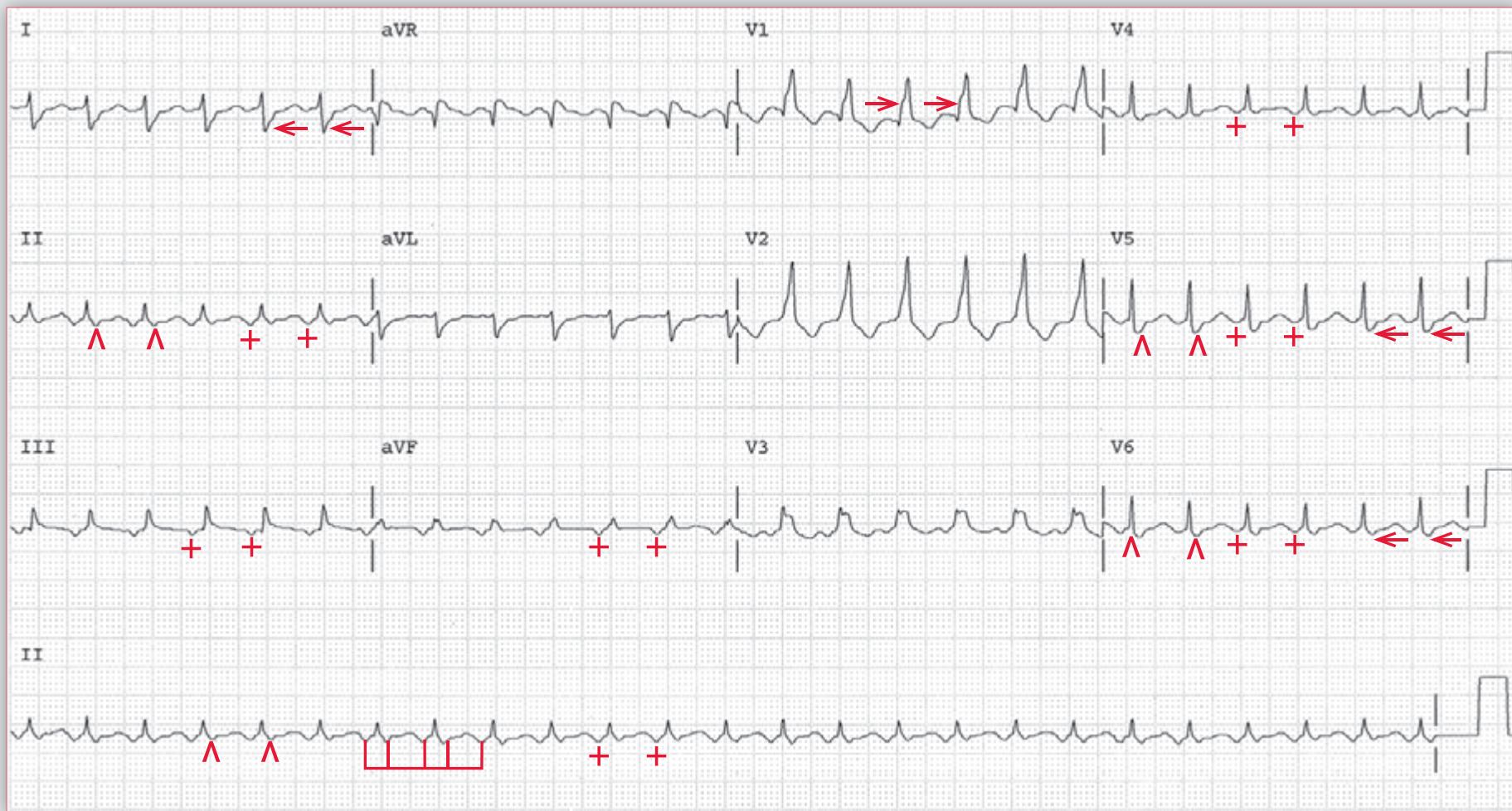
Before the clinician can arrive, the patient's symptoms resolve. The nurse obtains another ECG (ECG 16B).

What diagnosis is confirmed?

ECG 16B



Podrid's Real-World ECGs



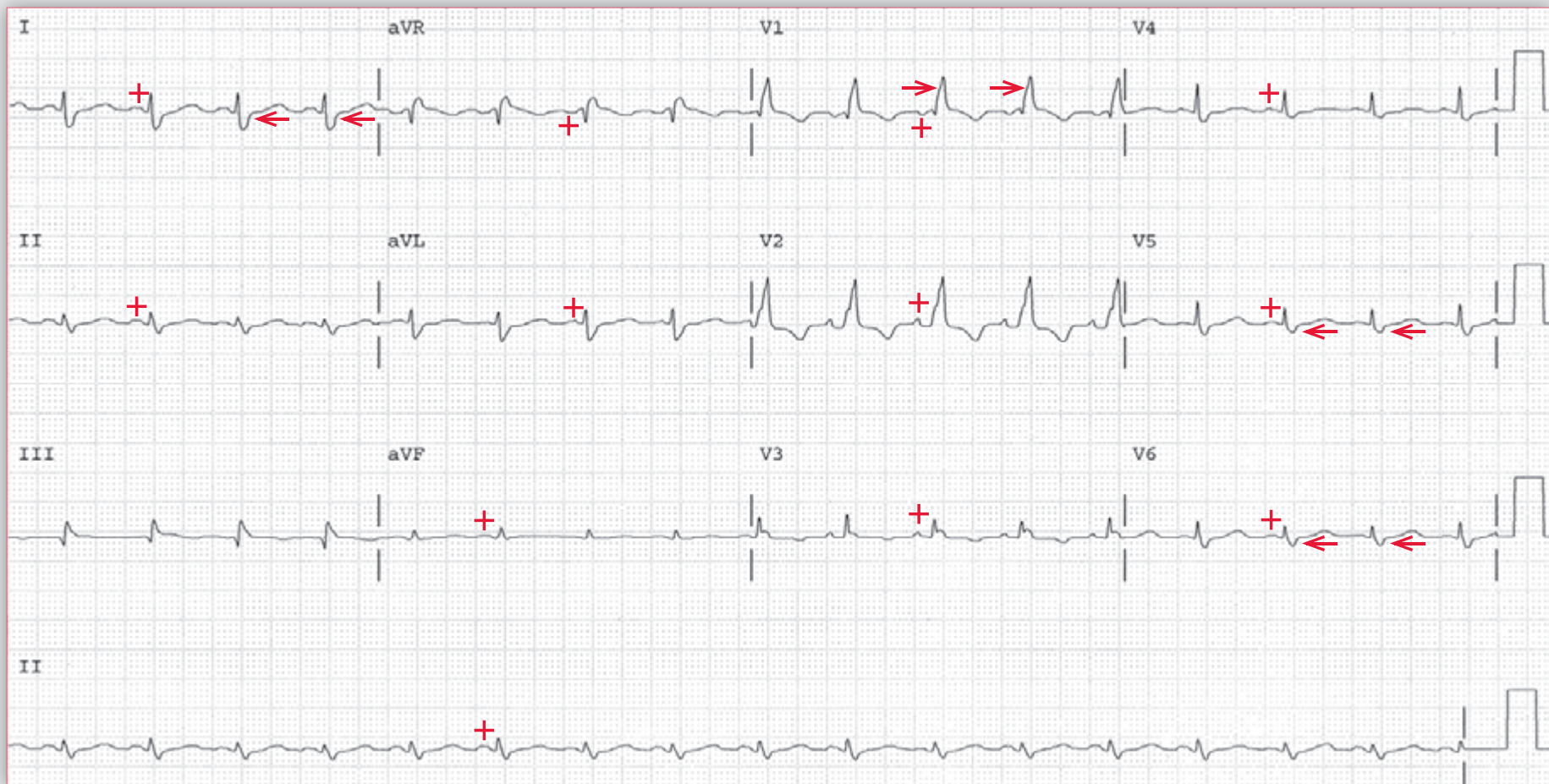
ECG 16A Analysis: Wide complex tachycardia, right bundle branch, long-RP tachycardia, atrial tachycardia, low voltage

ECG 16A shows a regular rhythm at a rate of 150 bpm. There is an atrial wave (+) seen before each QRS complex with a stable and very short PR interval (0.12 sec) (⌚) and a long RP interval (0.30 sec) (↔). These P waves are best seen in leads II, III, aVF, and V4–V6. This is a long PR tachycardia, and there are a number of etiologies for this type of tachycardia, including: sinus tachycardia, atrial tachycardia, ectopic junctional tachycardia, atrioventricular reentrant tachycardia, atypical atrioventricular nodal reentrant tachycardia (known as fast-slow), or atrial flutter with 2:1 AV nodal conduction. The P wave is negative in these leads; hence this is a rhythm originating in the atrial myocardium and not from the sinus node. Although there is a negative waveform after each QRS complex in leads II and V5–V6 (^), this is not a second atrial complex as the PP interval (↔) is not constant. Since there is only

one P wave for each QRS complex, which is negative in leads II, aVF, and V4–V6 and associated with a very short PR interval, this is not sinus tachycardia or atrial flutter. Of the other potential rhythms, the most likely etiology is atrial tachycardia.

The QRS complex duration is increased (0.14 sec) and there is a typical right bundle branch block morphology (broad R wave in V1 [→] and broad S wave in leads I and V5–V6 [←]). There is a normal axis between 0° and +90° (QRS positive in leads I and aVF). Also noted is low voltage throughout (QRS amplitude < 5 mm or little boxes in the limb leads and < 10 mm or little boxes in the precordial leads). The QT/QTc intervals are normal (280/440 msec and 240/370 msec when corrected for the prolonged QRS duration). *continues*

Podrid's Real-World ECGs



ECG 16B Analysis: Normal sinus rhythm, right bundle branch block, low voltage

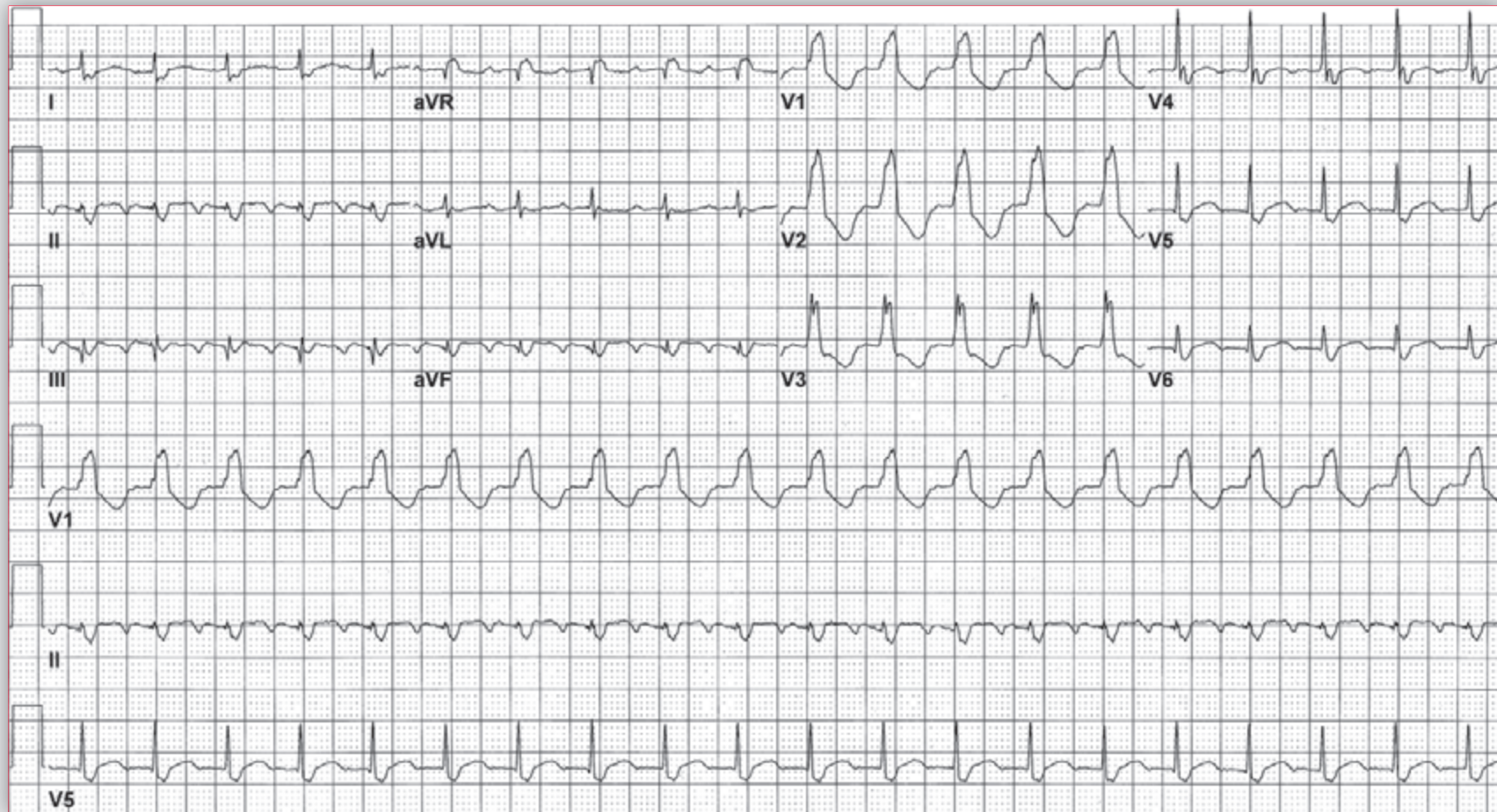
ECG 16B is from the same patient as ECG 16A. There is a regular rhythm at a rate of 98 bpm. The QRS morphology and axis are the same as ECG 16A, *ie*, there is a right bundle branch block morphology (RSR' in lead V1 [→] and broad S wave in leads I and V5–V6 [←]). There is a P wave (+) before each QRS complex with a stable PR interval (0.14 sec), and the P wave is positive in leads I, II, aVF, and V5–V6.

Hence this is a sinus rhythm with a baseline right bundle branch block morphology, indicating that the right bundle branch block in ECG 16A is not rate-related but is preexistent. Also noted is that the waveform after the QRS complex in leads II and V5–V6 is not a P wave, but is part of the QRS complex, *ie*, an S wave. ■

Core Case 17

A 68-year-old man is admitted to the cardiac care unit of a university hospital with an exacerbation of heart failure. Upon admission, he is noted to be tachycardic. As part of his evaluation, an ECG is obtained (ECG 17A).

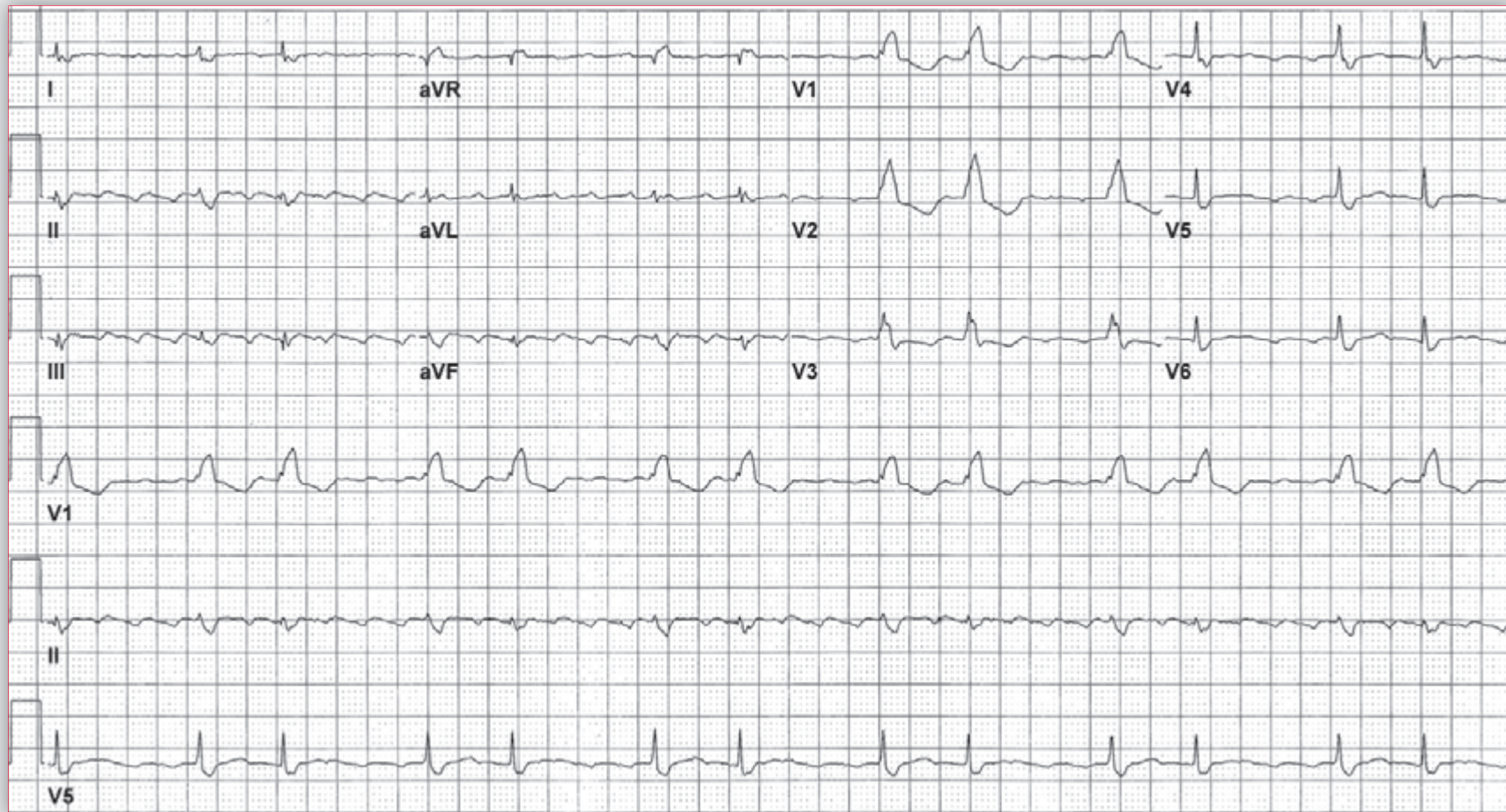
ECG 17A



Overnight, the patient's telemetry alarms.
The nurse obtains an ECG in response to a newly
manifested arrhythmia (ECG 17B).

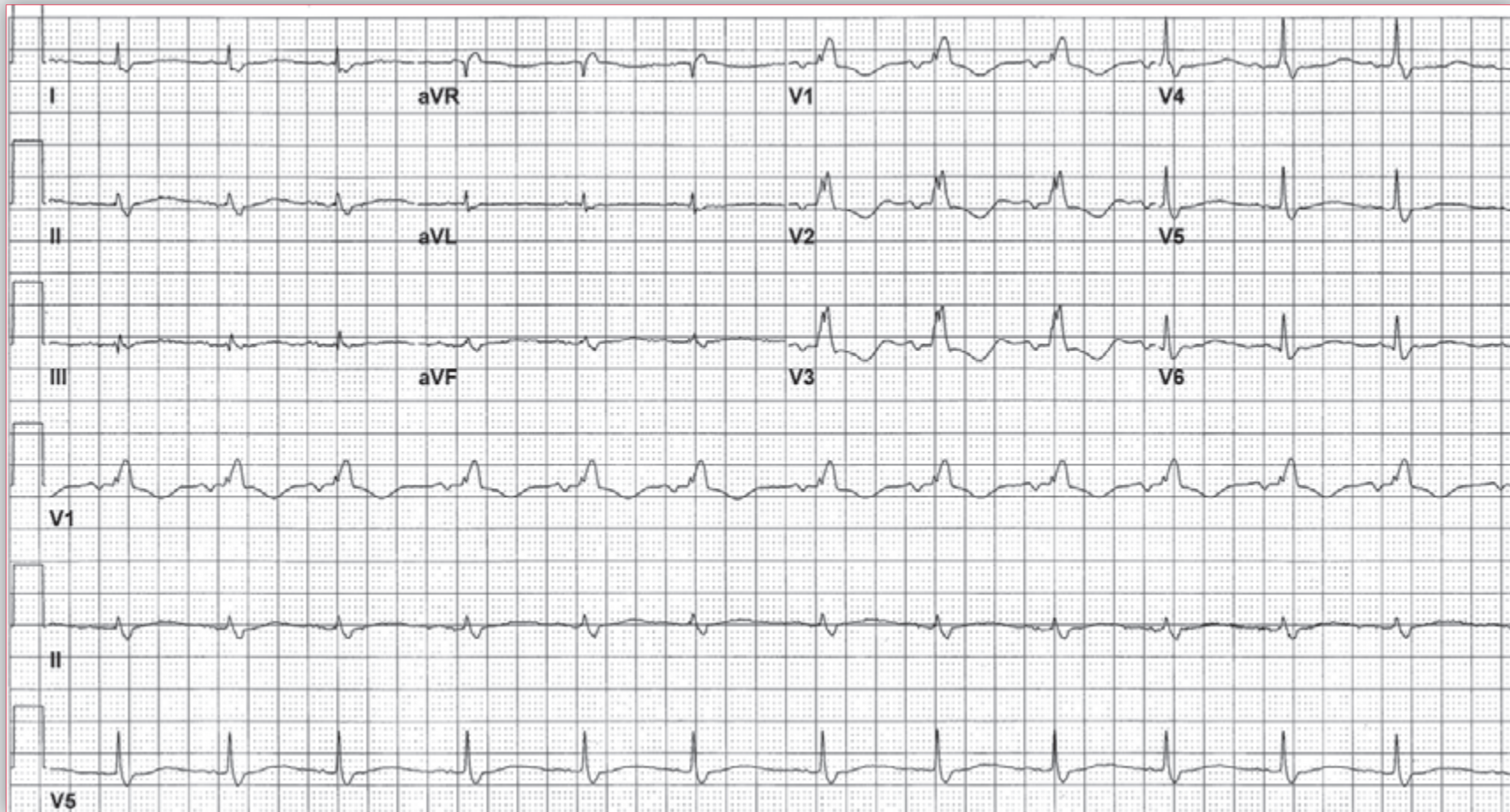
A follow-up ECG is obtained after a new
pattern is noted on telemetry (ECG 17C).

ECG 17B



Core Case 17

ECG 17C

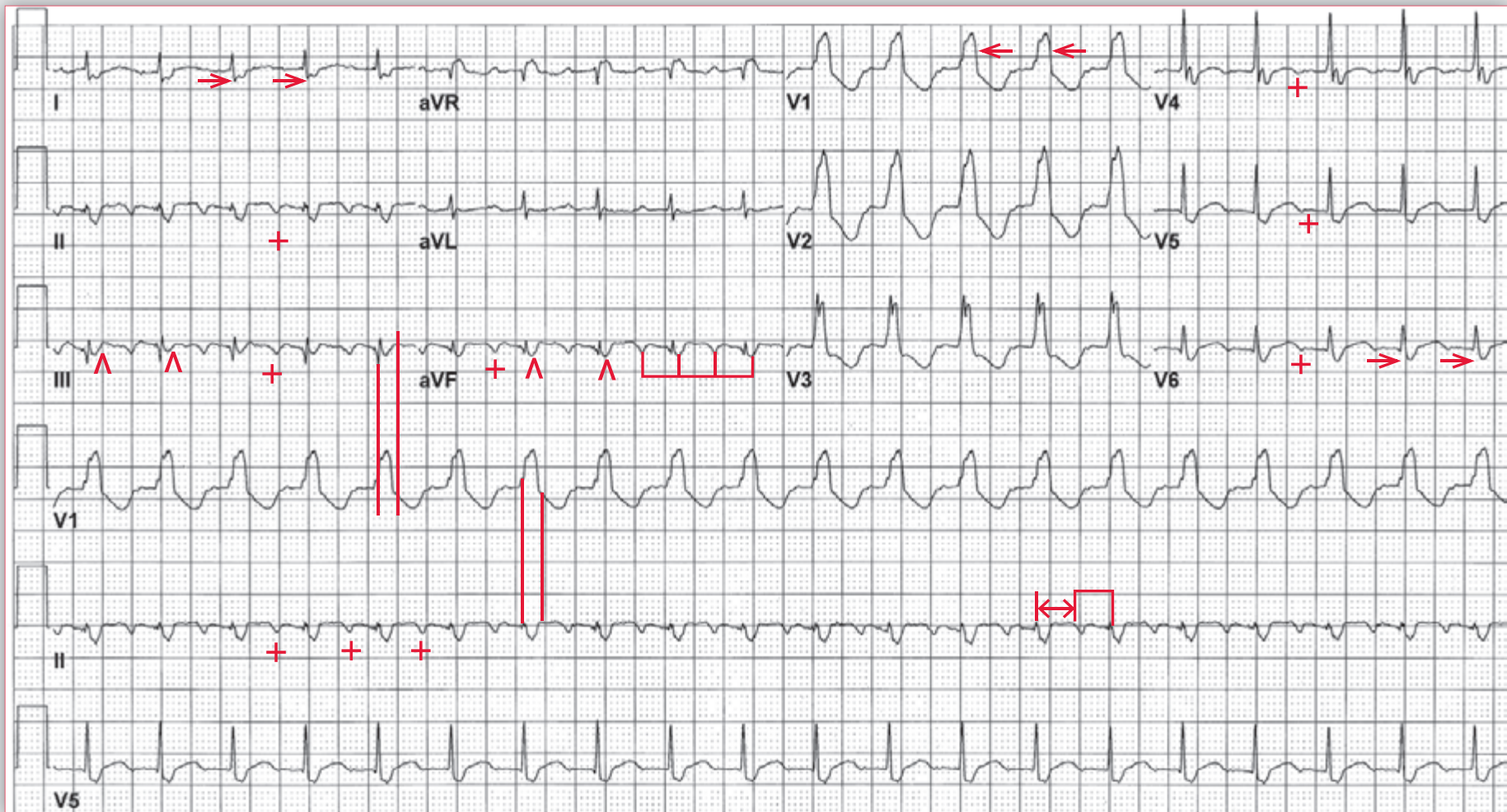


What arrhythmia does the tracing (ECG 17A) depict?

**What is the differential diagnosis
based on the tracing (ECG 17A)?**

What is your interpretation of the tracing (ECG 17B)?

What has been the interim development?



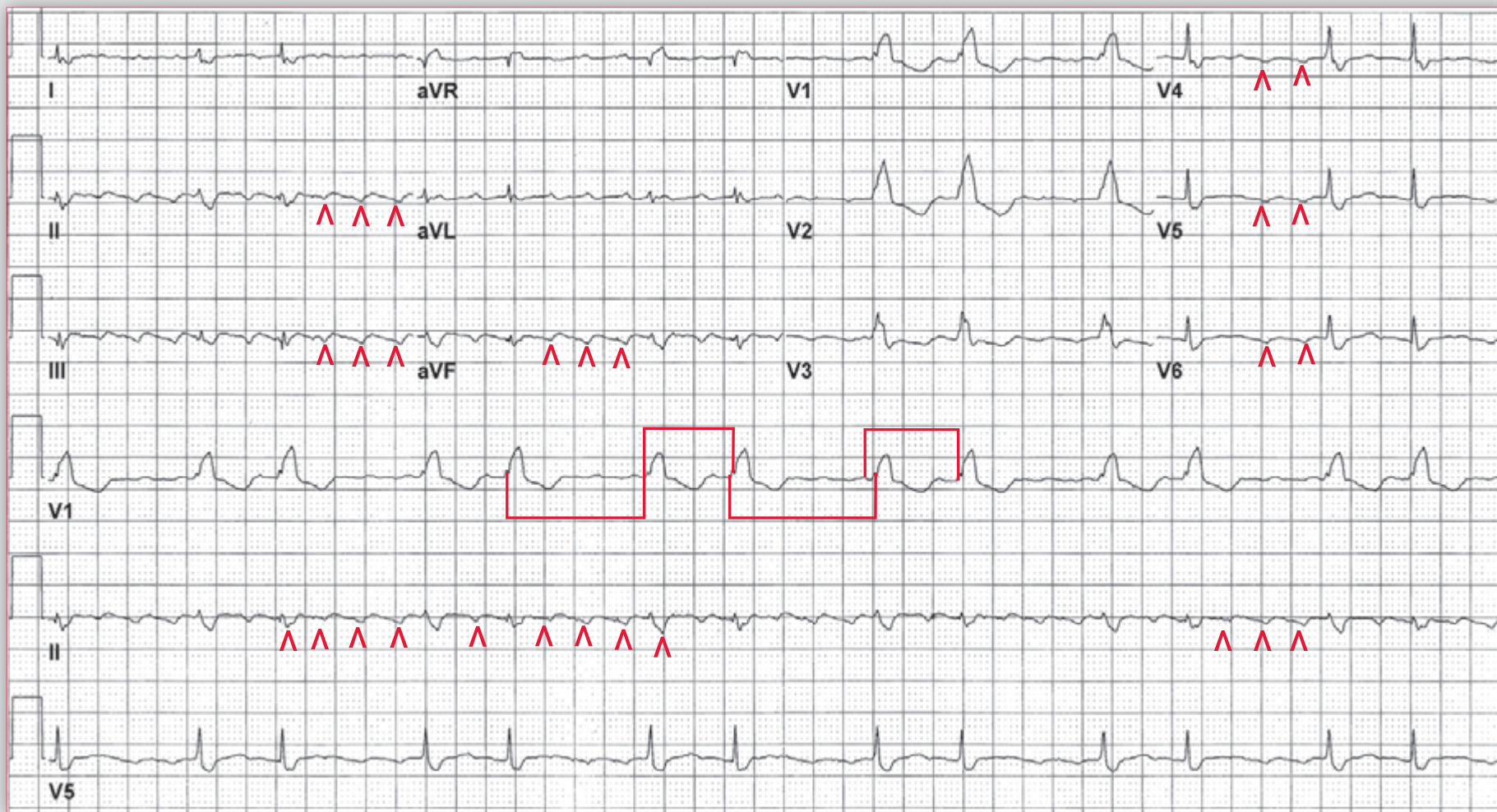
ECG 17A Analysis: Wide complex tachycardia with right bundle branch block, long-RP tachycardia, low voltage

ECG 17A shows a regular rhythm at a rate of 130 bpm. A P wave can be seen between each QRS complex, especially in leads II, III, aVF, and V4–V6 (+), and the PR interval (↔) is slightly longer (0.28 sec) than the RP interval (0.22 sec) (⌐); hence this is a long-RP tachycardia. The etiologies for a long-RP tachycardia include: sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, an ectopic junctional tachycardia, an atrioventricular reentrant tachycardia, or an atypical atrioventricular nodal reentrant tachycardia (AVNRT) known as fast-slow. The P wave is negative in the leads where it is seen (II, III, aVF and V4–V6). Therefore, the initial impulse is not originating from the sinus node, but rather from some focus within the atrial myocardium. There appears to be another negative waveform of a similar morphology immediately after the QRS complex (^), best seen in leads III and

aVF. The intervals between these waveforms is constant (⌐). If the waveform is a P wave, the PP interval is 260 bpm and the rhythm is atrial flutter with 2:1 AV block. However, it is not certain if this is another P wave or actually is part of the QRS complex, *ie*, an S wave, as when compared to the QRS complex width in lead V1 (⌐), it seems as if this waveform is part of the QRS complex. The QRS complexes are wide (0.16 sec) with a right bundle branch block morphology, *ie*, broad R wave in lead V1 (←) and a terminal S wave in leads I and V5–V6 (→). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). There is low voltage in the limb leads (QRS complex < 5 mm in each limb lead). The QT/QTc intervals are prolonged (320/470 msec) but are normal when the widened QRS complex is considered (260/370 msec).

continues

Podrid's Real-World ECGs



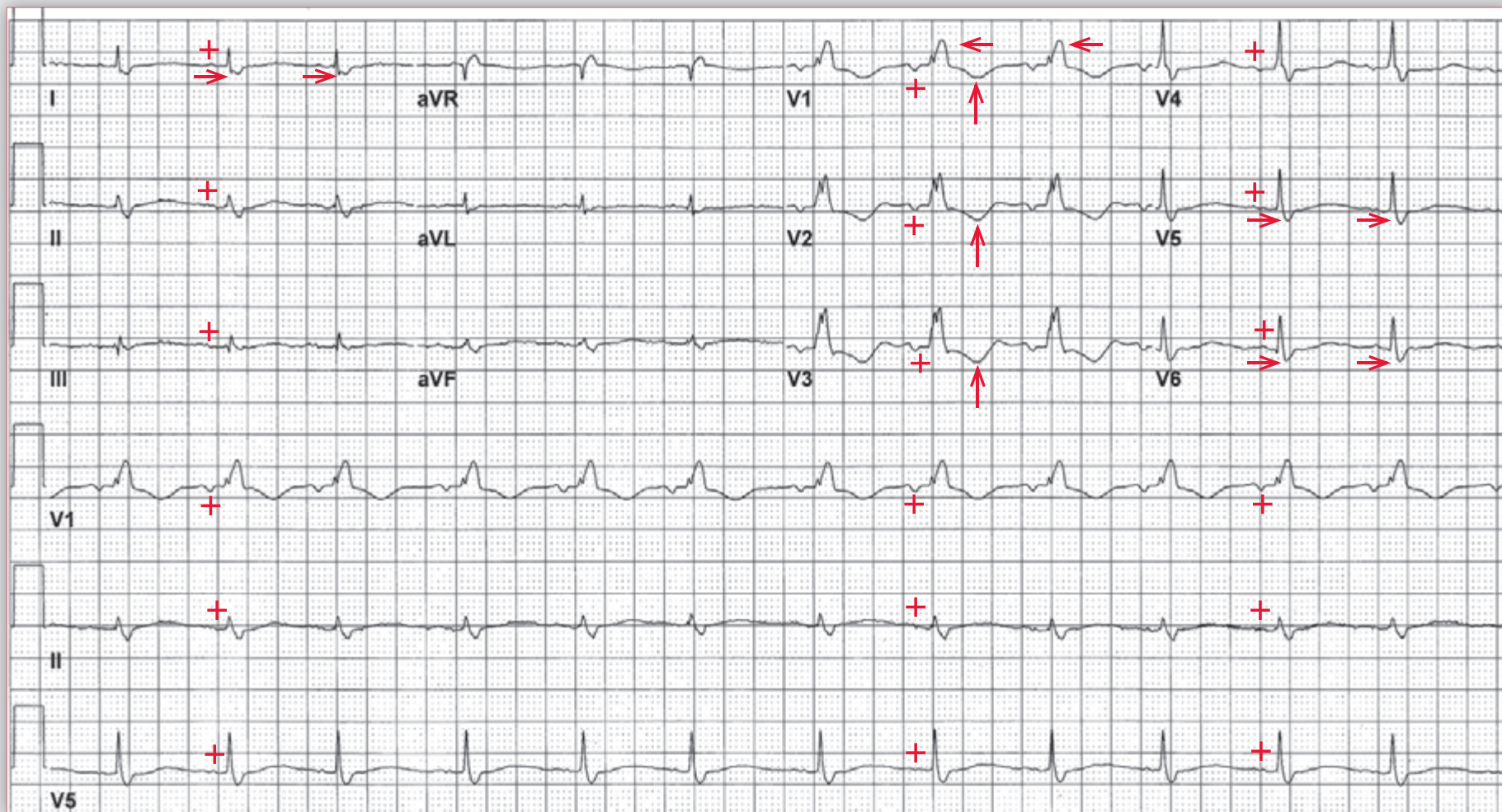
ECG 17B Analysis: Atrial flutter with variable block, right bundle branch block, low voltage

ECG 17B is from the same patient as ECG 17A. There is a regularly irregular rhythm with an average rate of 78 bpm. There are long (⏏) and short (⏏) intervals, all of which are the same, *ie*, group beating. The QRS complex width and morphology are identical to that in ECG 17A, *ie*, there is a right bundle branch block, low voltage in the limb leads and a normal axis. The QT/QTc intervals are the same as ECG 17A. There is obvious atrial activity seen, especially in leads II, III, aVF, and V4–V6 (^). The atrial waveforms are negative in leads II, aVF, and V4–V6. The atrial rate is 260 bpm and all the waveforms

have the same morphology, amplitude, and interval. Hence this is atrial flutter with variable block (2:1 and 4:1). The atrial rate of 260 bpm is twice the ventricular rate noted in ECG 17A, and the shorter RR intervals noted on this ECG are the same as the RR intervals noted in ECG 17A. This establishes the rhythm seen on this ECG as atrial flutter with 2:1 AV block. The right bundle branch block is preexistent and seen even when the RR interval are longer (slower heart rate). Hence it is not rate-related.

continues

Podrid's Real-World ECGs



ECG 17C Analysis: Normal sinus rhythm, right bundle branch block morphology, low voltage

ECG 17C is from the same patient as ECG 17A and ECG 17B, and is the patient's baseline ECG. There is a regular rhythm at a rate of 75 bpm. There is a P wave (+) before each QRS complex, with a constant PR interval of 0.16 second. The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complexes are wide (0.16 sec) and have the same morphology and axis as the QRS complexes seen in ECGs 17A and 17B. They have a right bundle branch block pattern with a tall R wave in lead V1 (←) and a

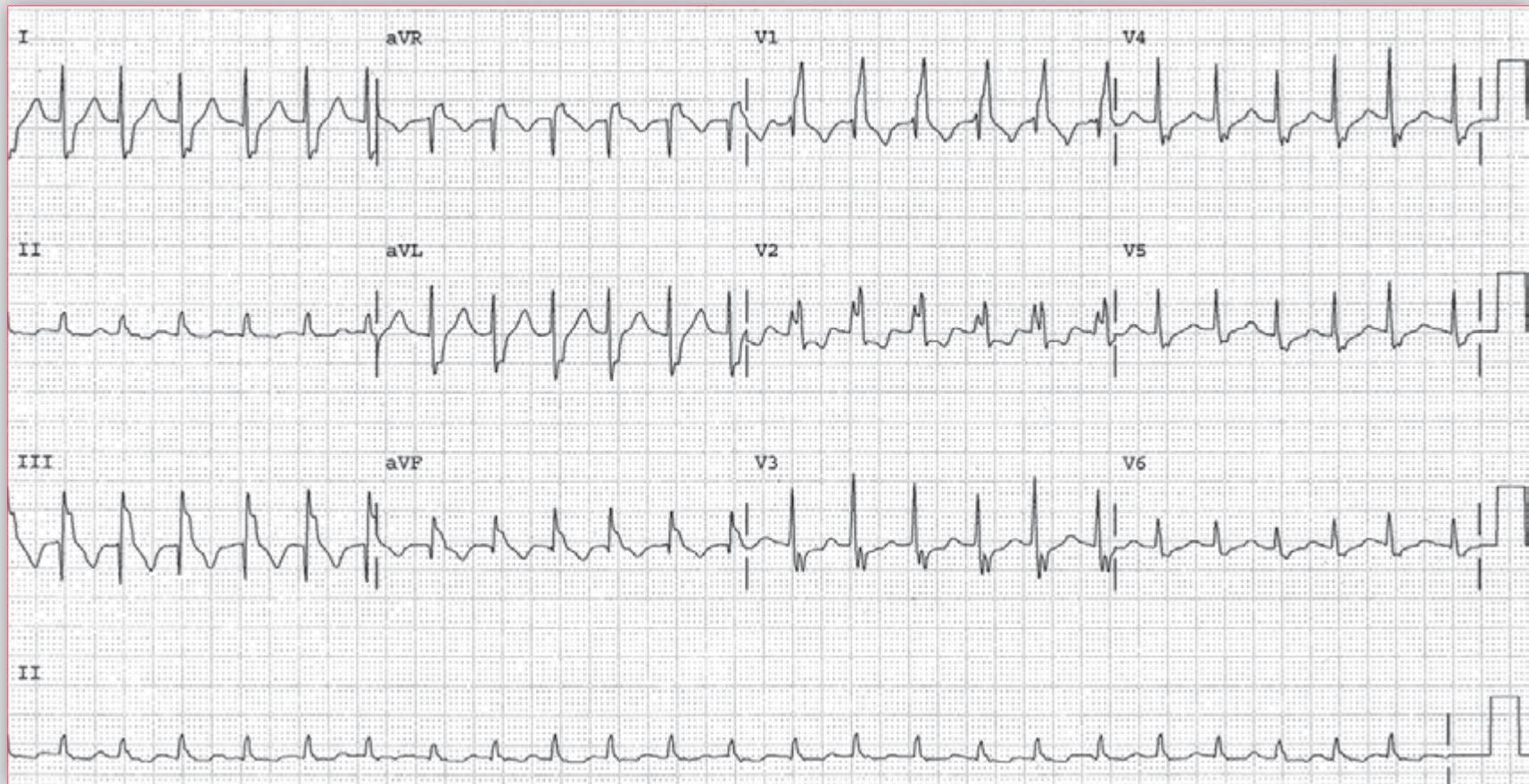
broad terminal S wave in leads I and V5–V6 (→). This further confirms the fact that the right bundle branch block is preexistent and is baseline for this patient; it is not rate-related. The QT/QTc intervals are prolonged (440/490 msec) but normal when the prolonged QRS complex duration is considered (360/400 msec). There are T-wave inversions in leads V1–V3 (↑); these are secondary to the right bundle branch block. ■

Core Case 18

A 59-year-old man is admitted to hospital for evaluation of acute-onset chest pain. During the night, he manifests a tachycardia that is evaluated with surface ECG (ECG 18A). The medical resident caring for the patient becomes alarmed by the tracing and pages her attending physician.

A follow-up ECG is obtained once the tachyarrhythmia resolves (ECG 18B).

ECG 18A

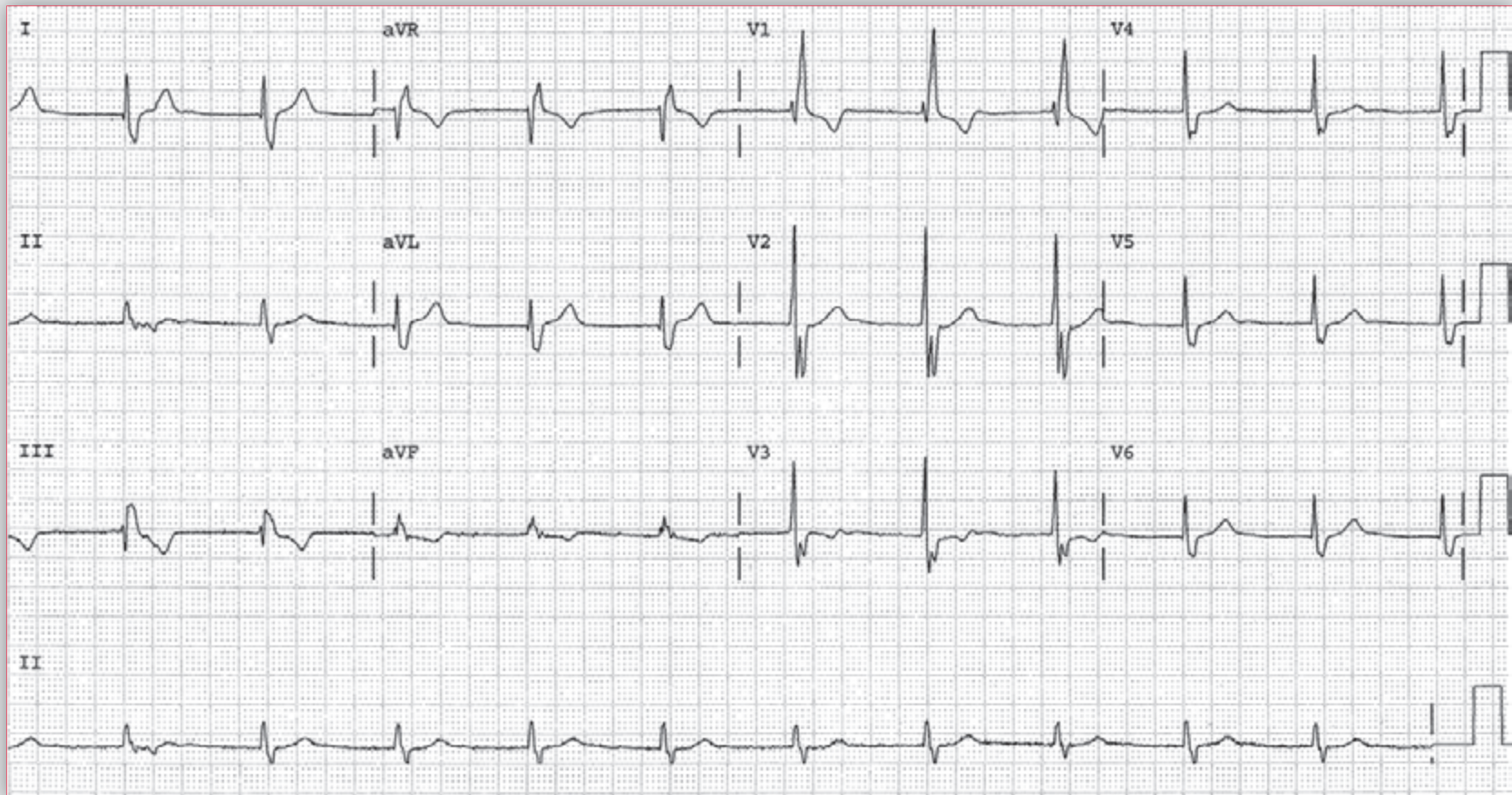


What is your interpretation of the ECG, and what possible diagnosis may have alarmed the resident?

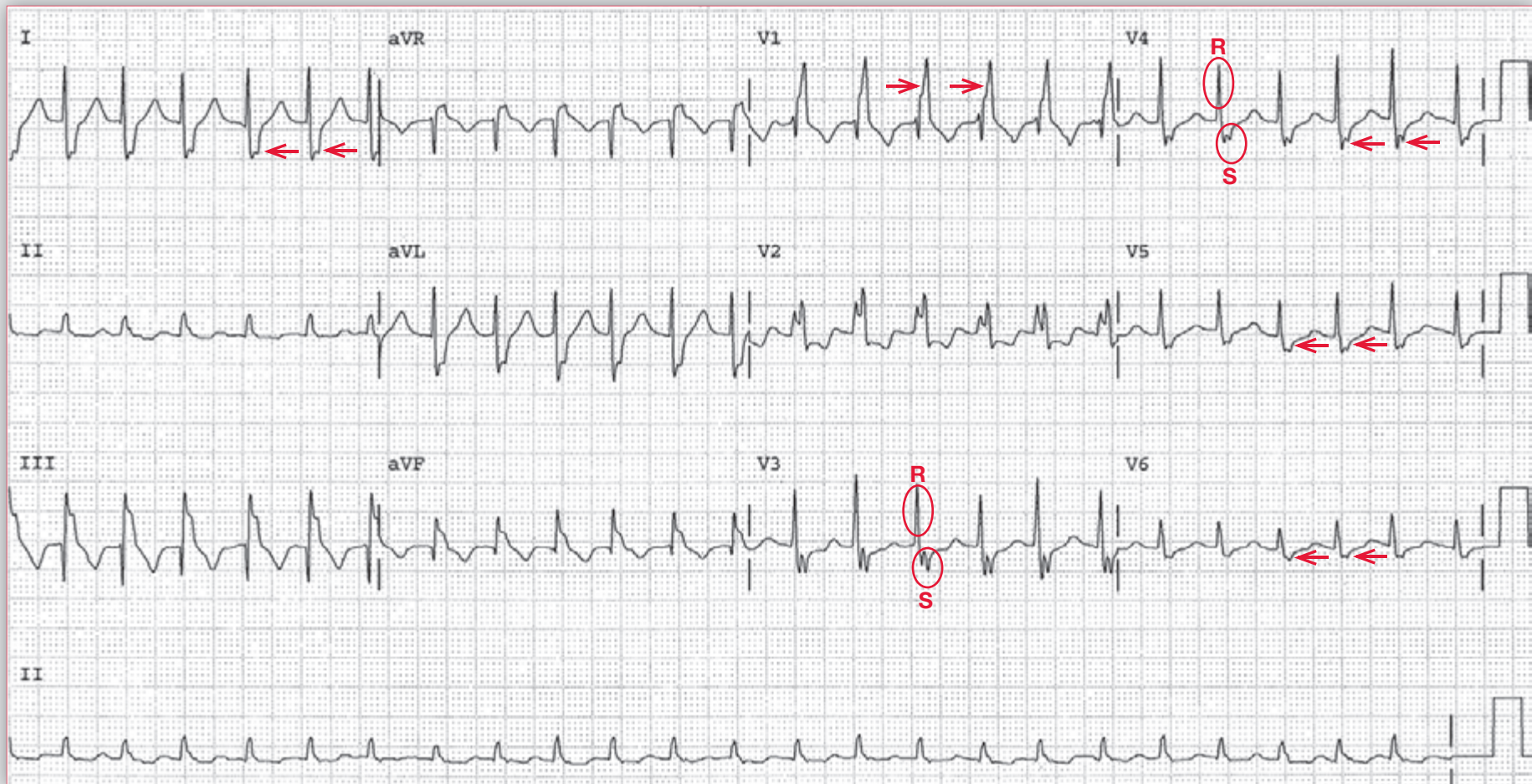
What is your interpretation of tracing B, and what further information does it provide?

Does it support or refute any possible diagnoses raised by tracing A?

ECG 18B



Podrid's Real-World ECGs



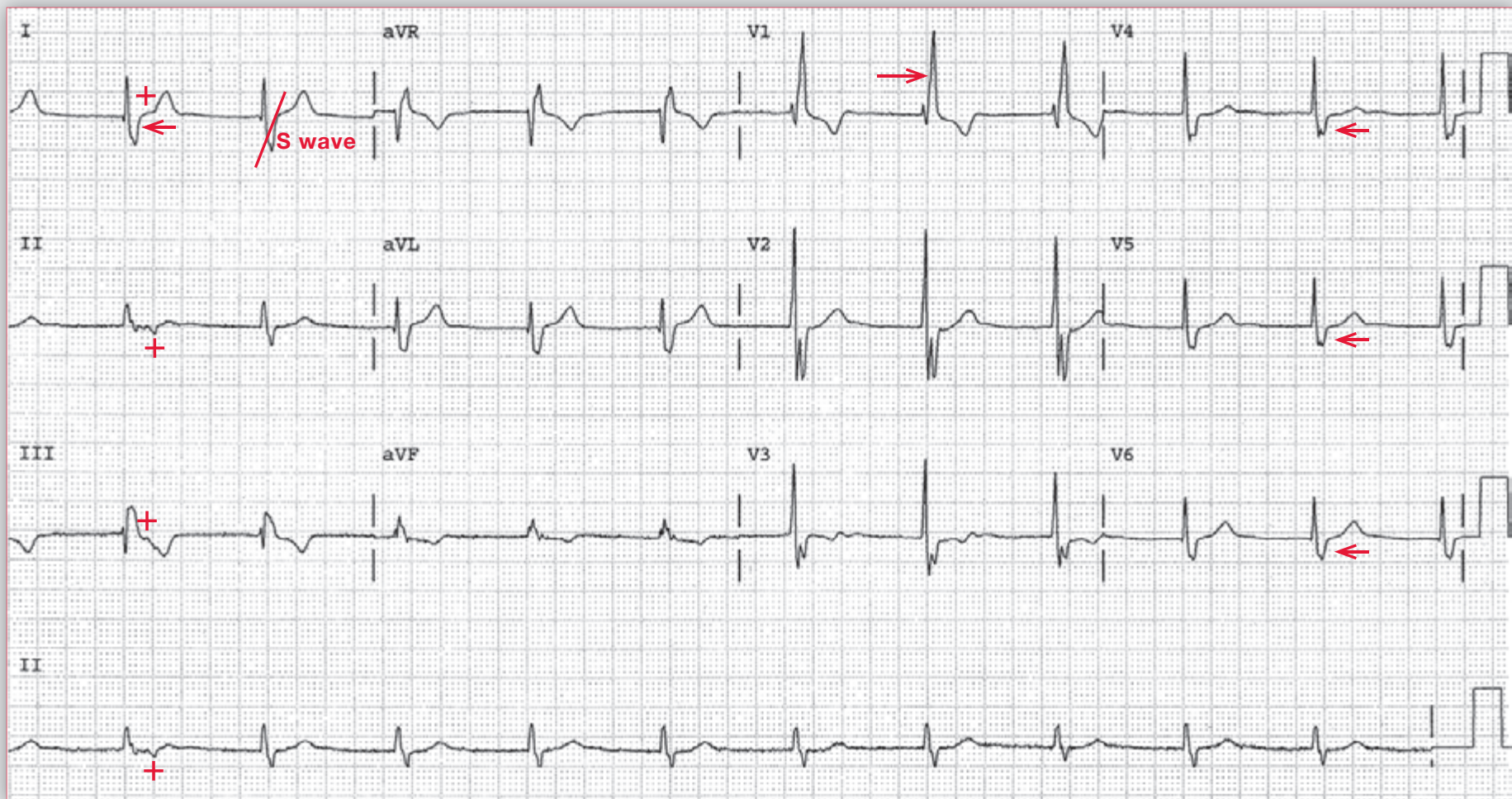
ECG 18A Analysis: Wide complex tachycardia, junctional tachycardia, right bundle branch block morphology

ECG 18A shows a regular rhythm at a rate of 144 bpm. There are no P waves seen before or after any of the QRS complexes. The QRS complex duration is increased (0.14 sec) and it has a right bundle branch block morphology. There is an RSR' in lead V1 (\rightarrow) and a broad terminal S wave in leads I and V4–V6 (\leftarrow). The axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are prolonged (320/490 msec) but are normal when the prolonged QRS complex duration is considered (280/420 msec). Although there is no atrial activity seen, the QRS morphology is typical for a

right bundle branch block. In addition, there is an RS complex in leads V3–V5 and it can be seen that the R wave is much narrower than the S wave ($R/S < 1$) and the R wave is < 100 msec in width. Hence this is a supraventricular tachycardia with aberration. As there are no P waves seen, this is termed a no-RP tachycardia, and it is a junctional tachycardia. The most likely etiologies for this are an atrioventricular nodal reentrant tachycardia (AVNRT) or an ectopic junctional tachycardia (although this usually is associated with a retrograde P wave).

continues

Podrid's Real-World ECGs



ECG 18B Analysis: Junctional rhythm, right bundle branch block morphology

ECG 18B is from the same patient as ECG 18A. There is a regular rhythm at a rate of 68 bpm. The QRS complexes are wide (0.14 sec) and have a right bundle branch block morphology with an RSR' in lead V1 (→) and a broad terminal S wave in leads I and V4–V6 (←). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). It is important not to include the S wave in lead I when determining axis. In this ECG, the S wave is broad and deep, giving an appearance that the QRS complex is negative in lead I. However, with the S wave is excluded, the QRS complex in this lead is positive. There are no P waves seen before or after any of the QRS complexes, except for the first QRS complex. A negative P wave is seen (+), particularly in leads I, II, and III. This is a junctional rhythm.

The QRS complex morphology and axis are identical to those seen in ECG 18A, confirming that the tachyarrhythmia in ECG 18A is supraventricular. Since the underlying rhythm is junctional, it is possible that this rhythm is an accelerated ectopic junctional tachycardia or an AVNRT. As the underlying rhythm is an ectopic junctional rhythm, the tachycardia is most likely due to any increase in the junctional rate. There is a right bundle branch block during the junctional tachycardia as well as during the slower junctional rhythm; the aberrancy is not rate-related but is preexistent. The QT/QTc intervals are prolonged (440/470 msec) but are normal when the prolonged QRS complex duration is considered (400/415 msec). ■

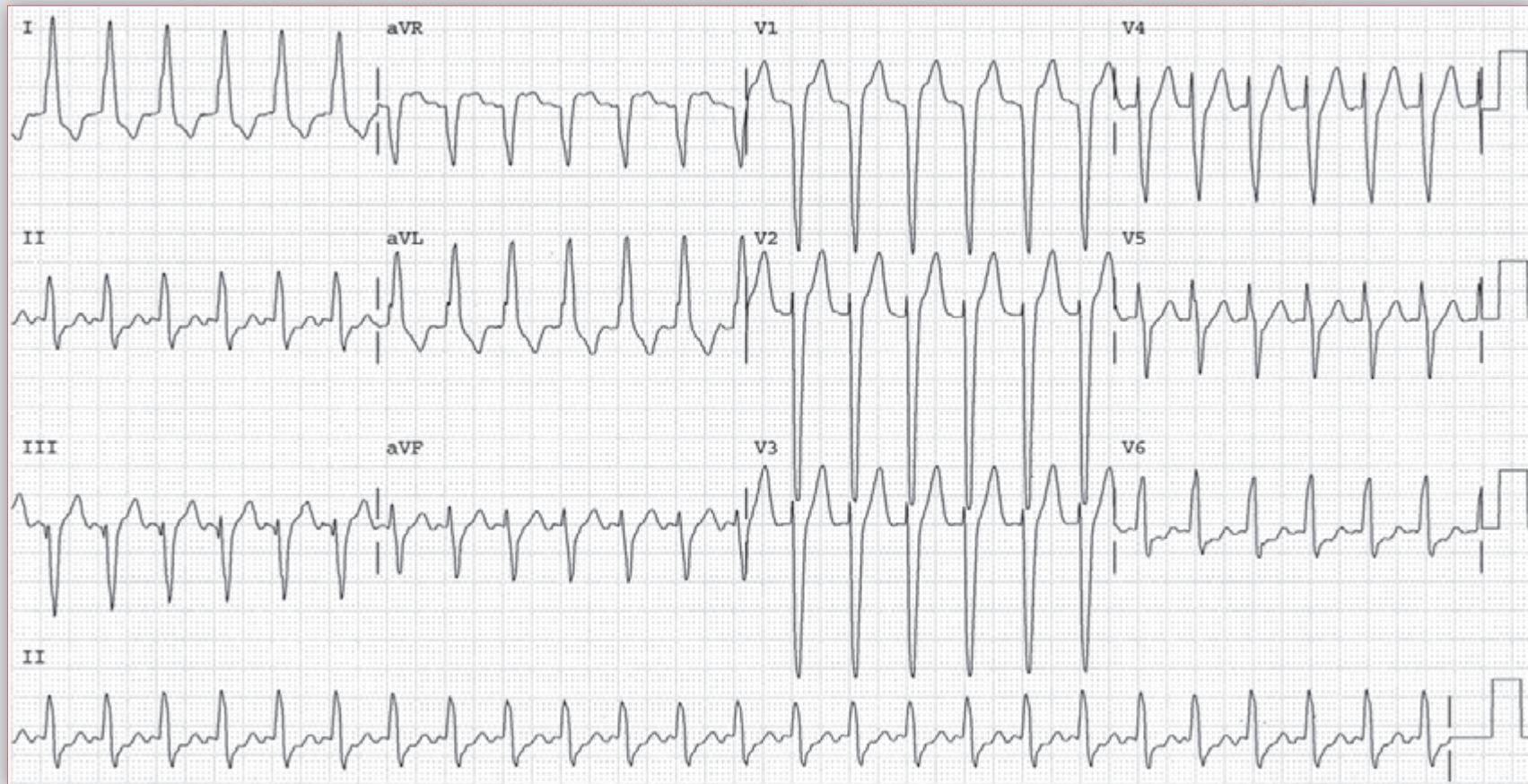
Core Case 19

A 78-year-old female is admitted to hospital after a fall. Her history raises the possibility of syncope. She described getting up from her chair in the study and, while walking to the kitchen, felt dizzy and unsteady, after which she fell to the ground. She cannot remember actually falling, but she does remember finding herself on the ground

with a headache, and she was able to dial 911 for assistance. She continues to remain lightheaded.

Her exam is notable for ecchymoses over the right orbit. Her cardiac impulse is accelerated, but heart sounds are normal. An S4 gallop is noted, as is a 2/6 crescendo-decrescendo murmur at the upper sternal border. Her extremities, in

ECG 19A



particular the palms of her hands, are without injury.

Her triage vital signs are notable for a rapid heart rate and normal blood pressure. An ECG is obtained in the emergency department (ECG 19A).

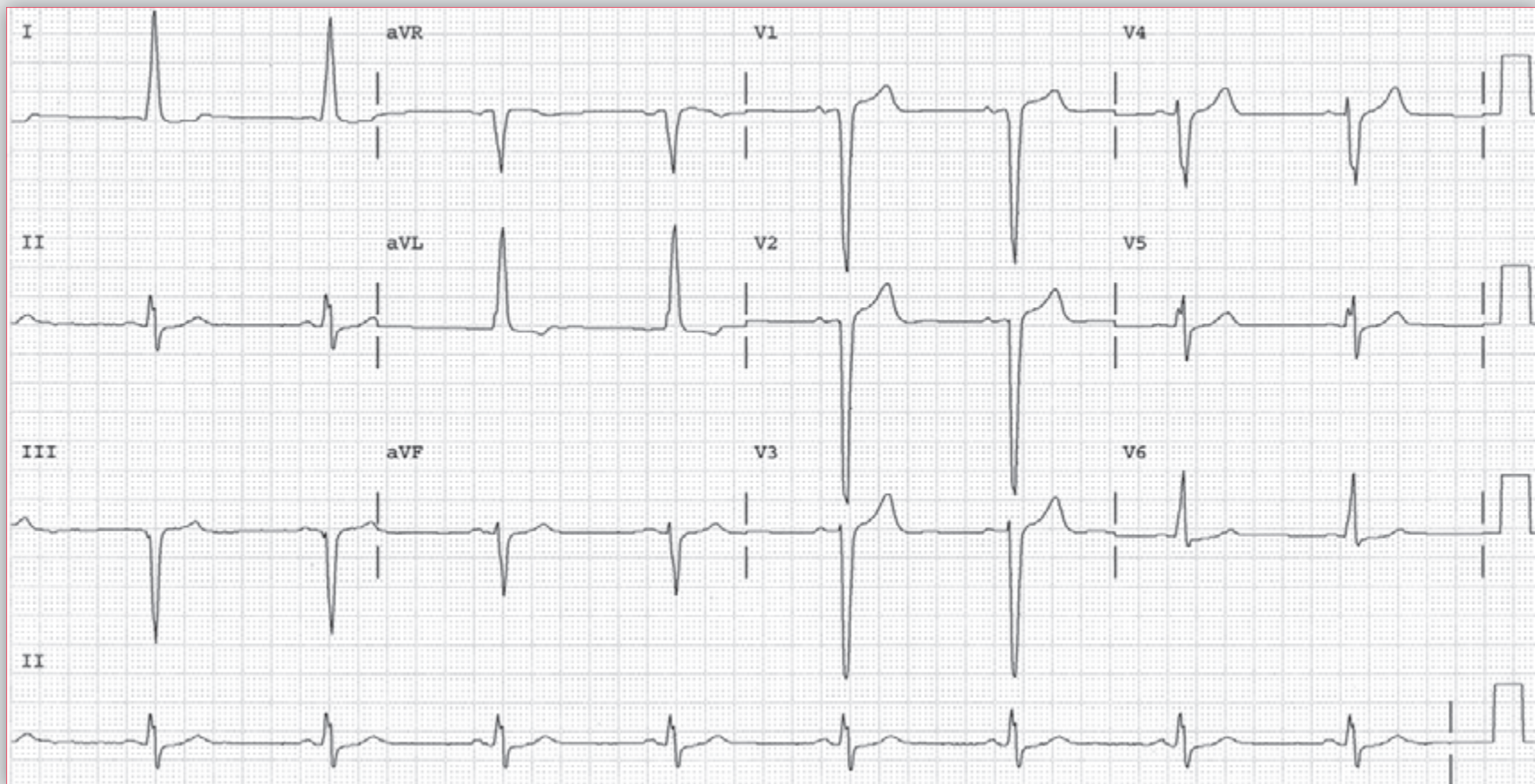
Upon arrival to the medical ward, her heart rate has slowed. A follow-up tracing is obtained (ECG 19B).

What is your interpretation of the tracing (ECG 19A)?

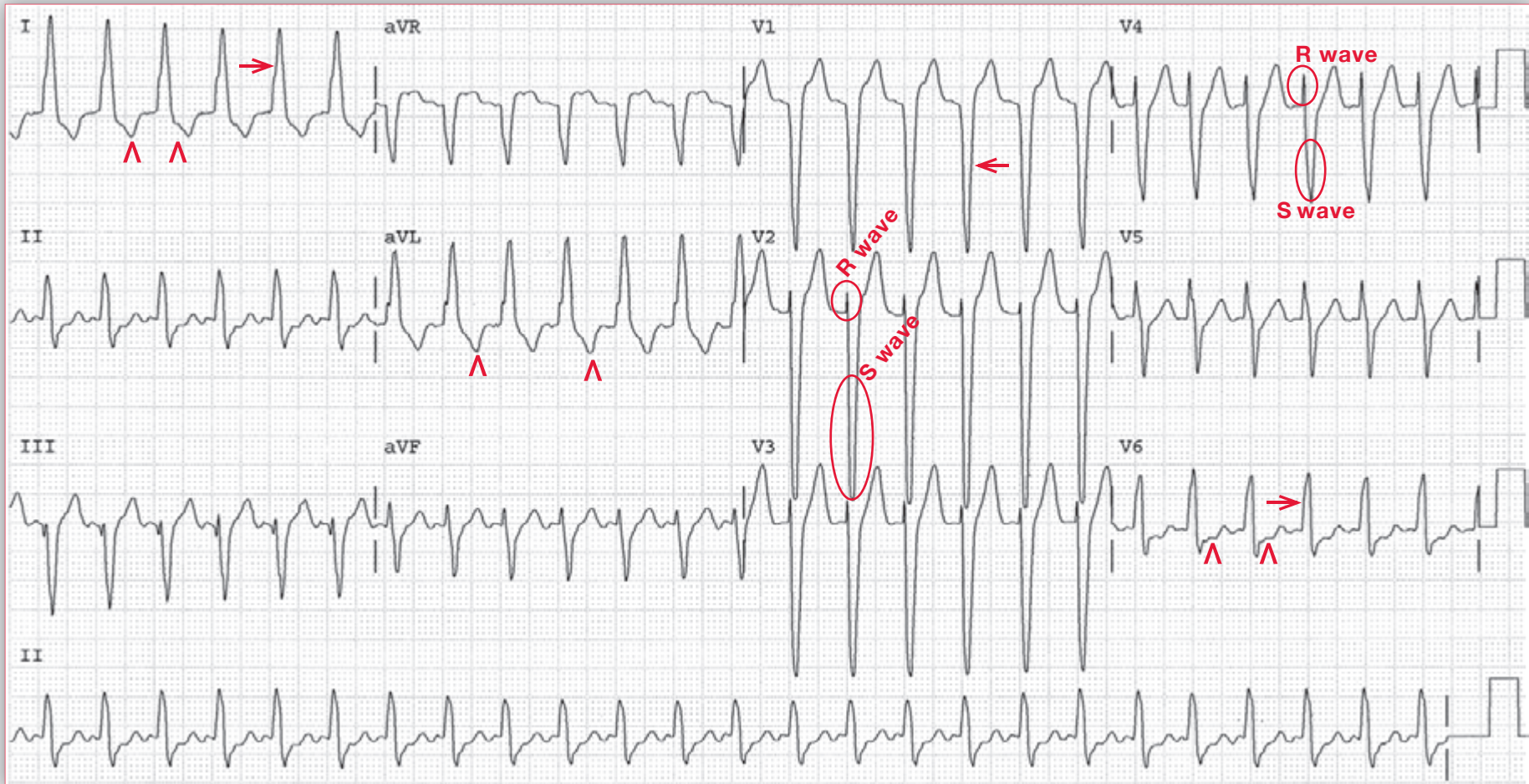
What arrhythmic diagnosis is suggested in comparing the tracings (ECG 19A and ECG 19B)?

How might this explain her presentation?

ECG 19B



Podrid's Real-World ECGs



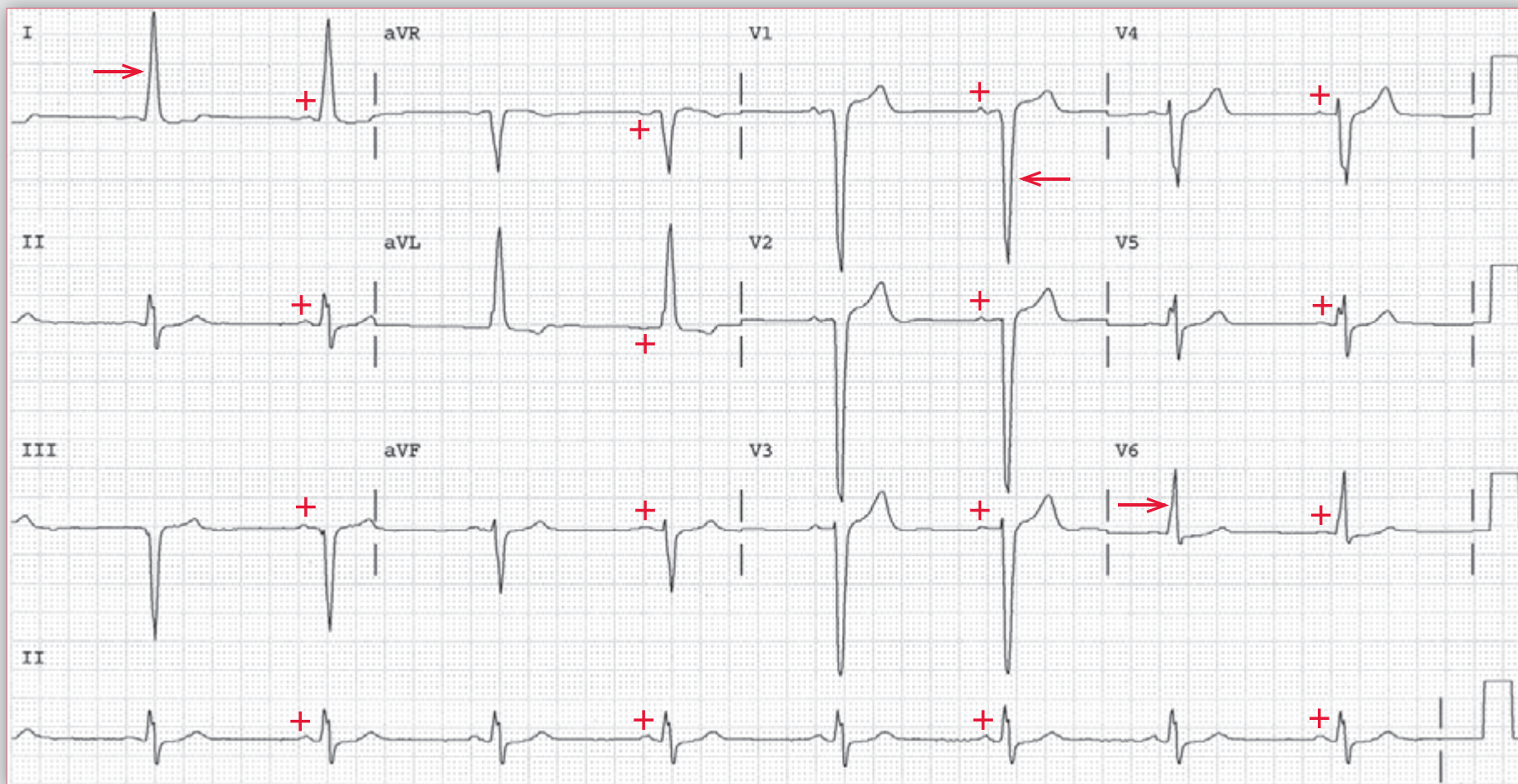
ECG 19A Analysis: Wide complex tachycardia due to an atrioventricular nodal reentrant tachycardia, left bundle branch block morphology

ECG 19A shows a regular rhythm at a rate of 150 bpm. There are no P waves seen before or after any of the QRS complexes. The QRS complex duration is increased (0.16 sec), and hence this is a wide complex tachycardia, which may be either ventricular tachycardia or a supraventricular tachycardia with aberration. The morphology is that of a typical left bundle branch block with a tall, broad R wave in lead I (→) and V6 and a deep QS complex in lead V1 (←). There are ST-T wave changes seen in leads I, aVL, and V6 (Λ). If this is a left bundle branch block, then these are secondary to the left bundle branch block. If this is a ventricular tachycardia, then the T-wave abnormalities reflect the presence of a ventricular complex. The axis is leftward between 0° and -30° (positive QRS complex in leads I and II and negative in

lead aVF). The QT/QTc intervals are prolonged (320/506 msec) but are normal when the prolonged QRS complex duration is considered (260/400 msec). It is not clear from this ECG if this is a supraventricular tachycardia or a ventricular tachycardia. However, there are R/S complexes in leads V2–V4, the R wave is wider than the S wave ($R/S < 1$), and the R wave is less than 100 msec. As the initial forces are normal, while it is the terminal portion of the QRS complex that is wide, this morphology is consistent with a supraventricular tachycardia with a left bundle branch block aberrancy. The absence of recognizable P waves establishes this as a no-RP tachycardia and the most common arrhythmia presenting in this fashion is an atrioventricular nodal reentrant tachycardia.

continues

Podrid's Real-World ECGs



ECG 19B Analysis: Sinus bradycardia, left bundle branch morphology

ECG 19B is from the same patient as ECG 19A. There is a regular rhythm at a rate of 50 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complexes are wide (duration = 0.16 sec) and have a typical left bundle branch block morphology with a broad R wave in leads I and V6 (→) and a deep QS wave in lead V1 (←). The axis is leftward between 0° and –30° (positive QRS complex in leads I and II and negative in lead aVF). The QT/QTc intervals are normal (480/440 msec and 420/390 msec when the prolonged QRS complex duration is considered). The QRS complexes in this ECG have the same morphology, width, axis, and QT interval as those seen in ECG 19A, thus, further confirming that the rhythm in ECG 19A is supraventricular and, therefore, an atrioventricular nodal reentrant tachycardia with aberration. The left bundle branch block is not rate-related as it is present at baseline.

A supraventricular tachycardia itself does not usually cause syncope, although at its very onset there may be failure of appropriate adjustment of vasomotor tone (mediated by baroreceptors) with the development of significant and transient hypotension. This may be associated with transient loss of consciousness. In addition, this patient has a murmur suggestive of aortic stenosis, which along with the rapid ventricular rate may be associated with hemodynamic changes resulting in syncope. The continued tachyarrhythmia may be associated with continued hypotension with persistent lightheadedness. ■

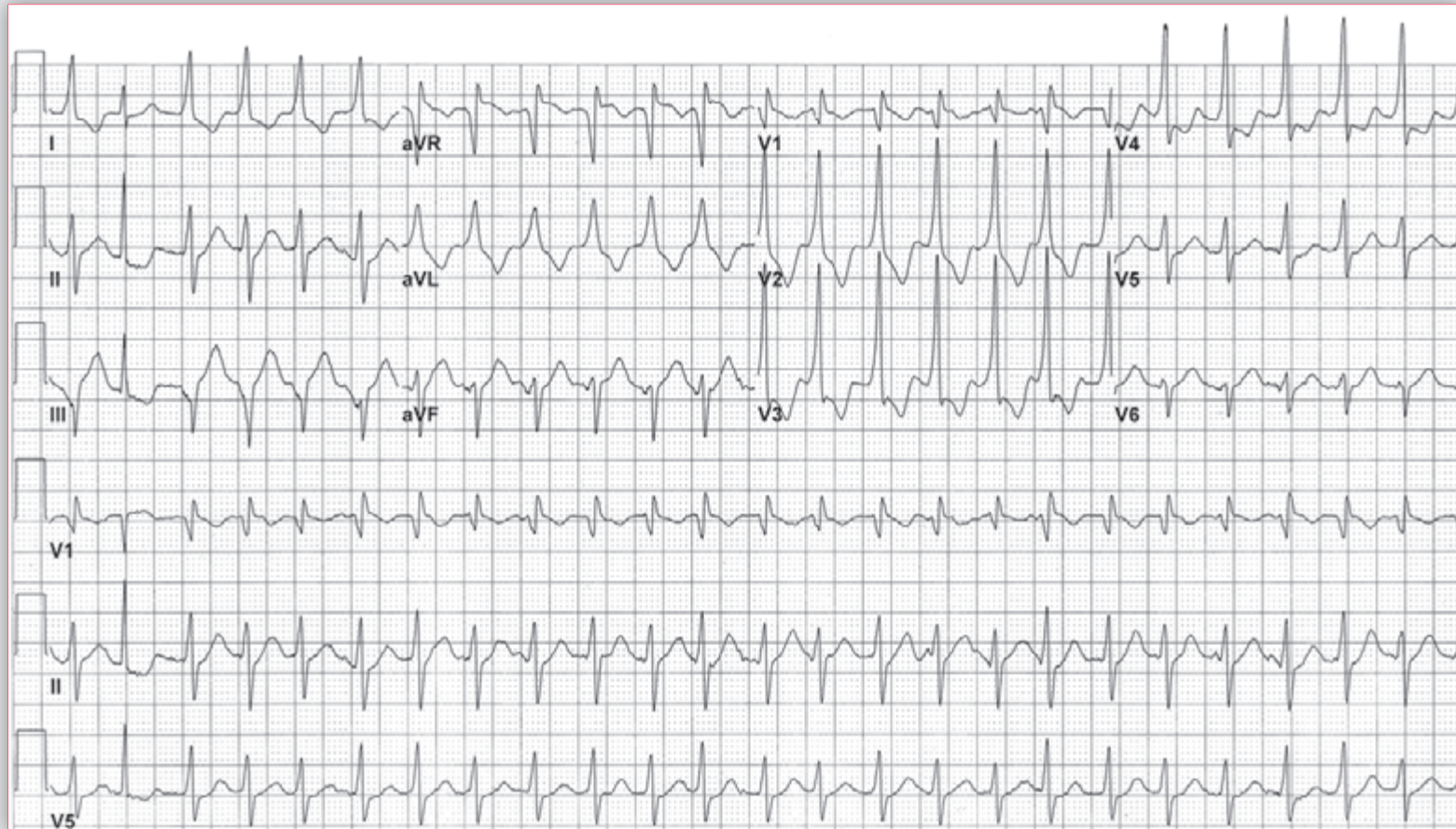
Core Case 20

A 22-year-old man presents to his primary doctor with a complaint of having a “fast heartbeat.” He states he has noted episodes of this on and off for the past several months. He generally notices this symptom when lying down at night to sleep. The sensation will start abruptly and end just as abruptly several minutes later.

He denies associated symptoms such as dyspnea, chest discomfort, or presyncope.

Physical examination reveals a well-developed male without distress. His physical examination is normal, as is his cardiac examination. Shortly after being seen, he complains of his typical fast heartbeat. At the time, his blood pressure is

ECG 20A



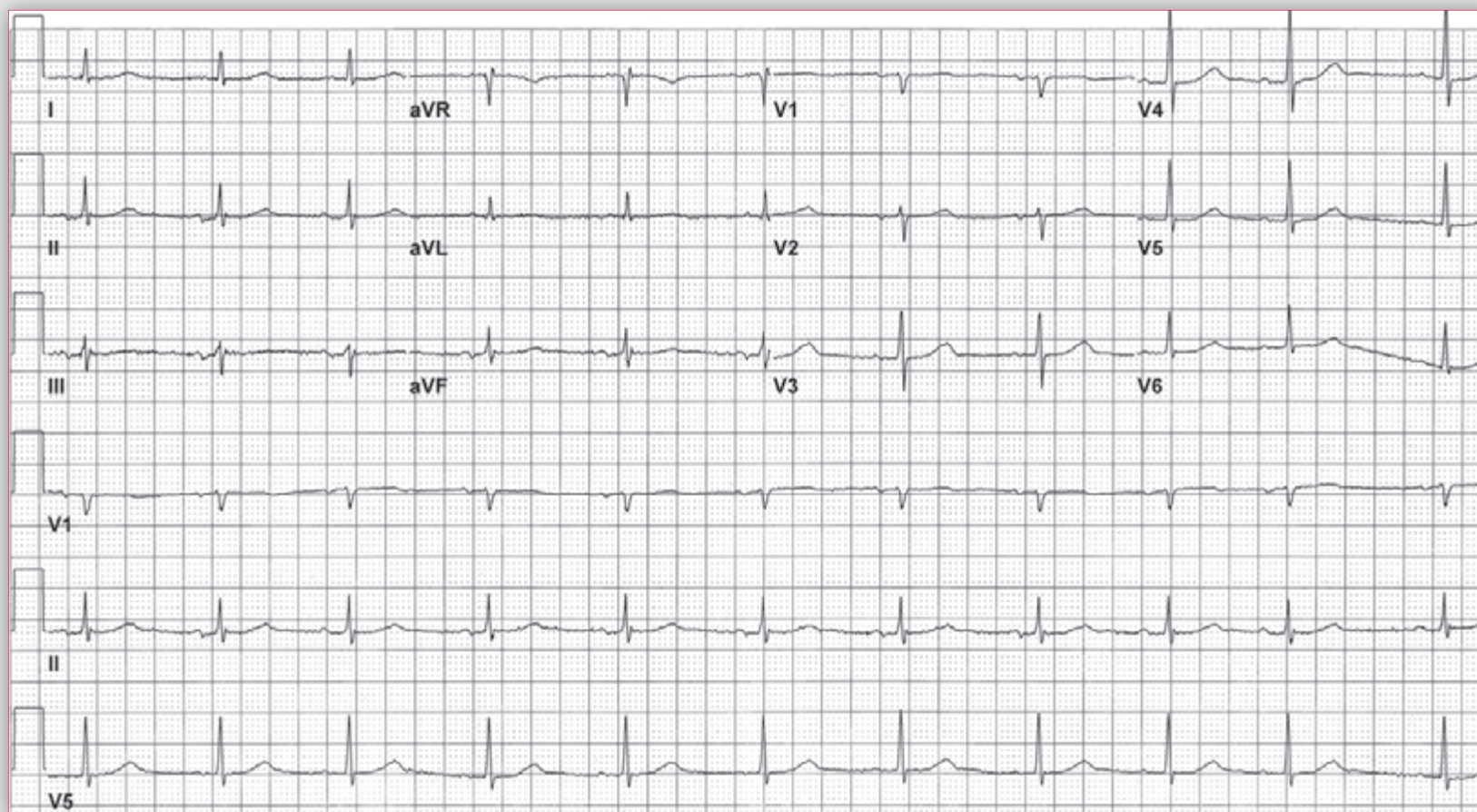
slightly lower than before, and examination of his heart reveals a rapid rate with heart sounds that are not regular.

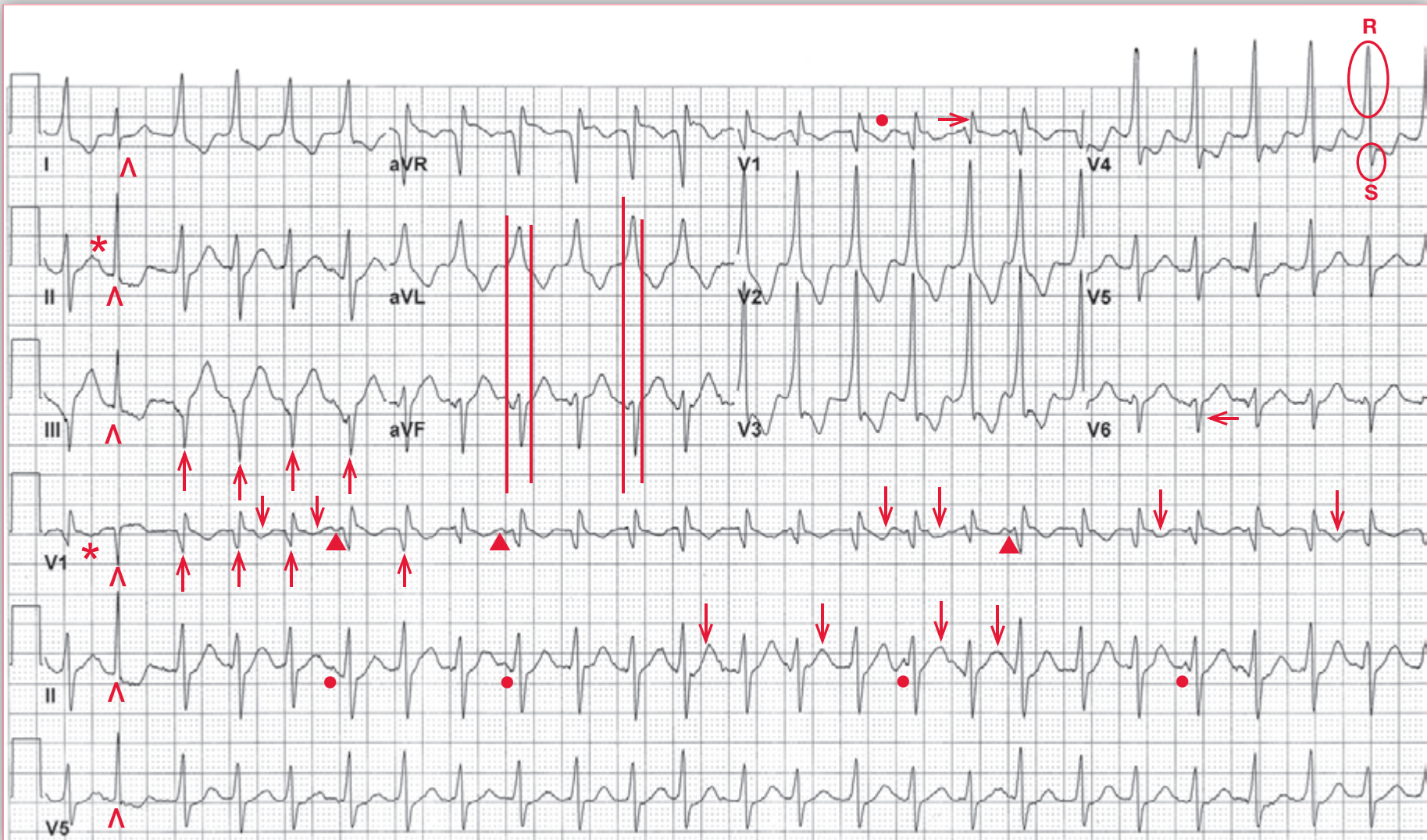
You obtain an ECG to evaluate the physical exam finding (tracing A). You compare this tracing (ECG 20A) to an ECG the patient received as part of a physical exam for competitive sports 2 years prior (ECG 20B).

What is the arrhythmic diagnosis?

What diagnosis is confirmed?

ECG 20B





ECG 20A Analysis: Wide complex tachycardia, AV dissociation, capture beat, ventricular tachycardia (fascicular tachycardia)

ECG 20A shows a regular rhythm at a rate of 148 bpm. The QRS complexes are wide, with a duration of 0.14 second. Although the QRS complexes resemble a right bundle branch block with an R wave in lead V1 (→) and a narrow S wave in lead V6 (←), there is no S wave in lead I and the morphology is not typical for a right bundle branch block. In addition it does not have a morphology of a left bundle branch block. The axis is leftward, approximately -30° (QRS complex positive in lead I, negative in lead aVF and biphasic in lead II). There is one narrow QRS complex noted (0.08 sec) (^). Before this QRS complex there is a P wave present, best seen in lead II and the V1 rhythm strip (*); the PR interval is 0.18 sec. There are other P waves seen (●, ▼), but none of them have a constant relationship to the QRS complexes, *ie*, they are dissociated. These P waves are most obviously seen in leads V1 and II rhythm strips, for example before the sixth, ninth, sixteenth, twenty-first QRS complexes (▼) in lead V1 and before the sixth, ninth, eighteenth complex in lead II (●). In addition there are subtle differences in the ST-T wave morphologies (↓) (which represent either superimposed P waves or changes in repolarization). There are also subtle differences in QRS complex morphology (↑). Therefore, this wide complex tachycardia has AV dissociation, which is characteristic of ventricular tachycardia. The narrow complex (^) is a captured or Dressler beat, which is a feature of AV dissociation. The subtle differences in ST-T waves and QRS complex morphology are characteristic of ventricular tachycardia. This is due to the fact that ventricular activation does not occur via the normal Purkinje pathway, but rather is directly through the ventricular muscle. Therefore, there may be changes in the activation and repolarization sequence due to local characteristics of the ventricular myocardium, resulting in the

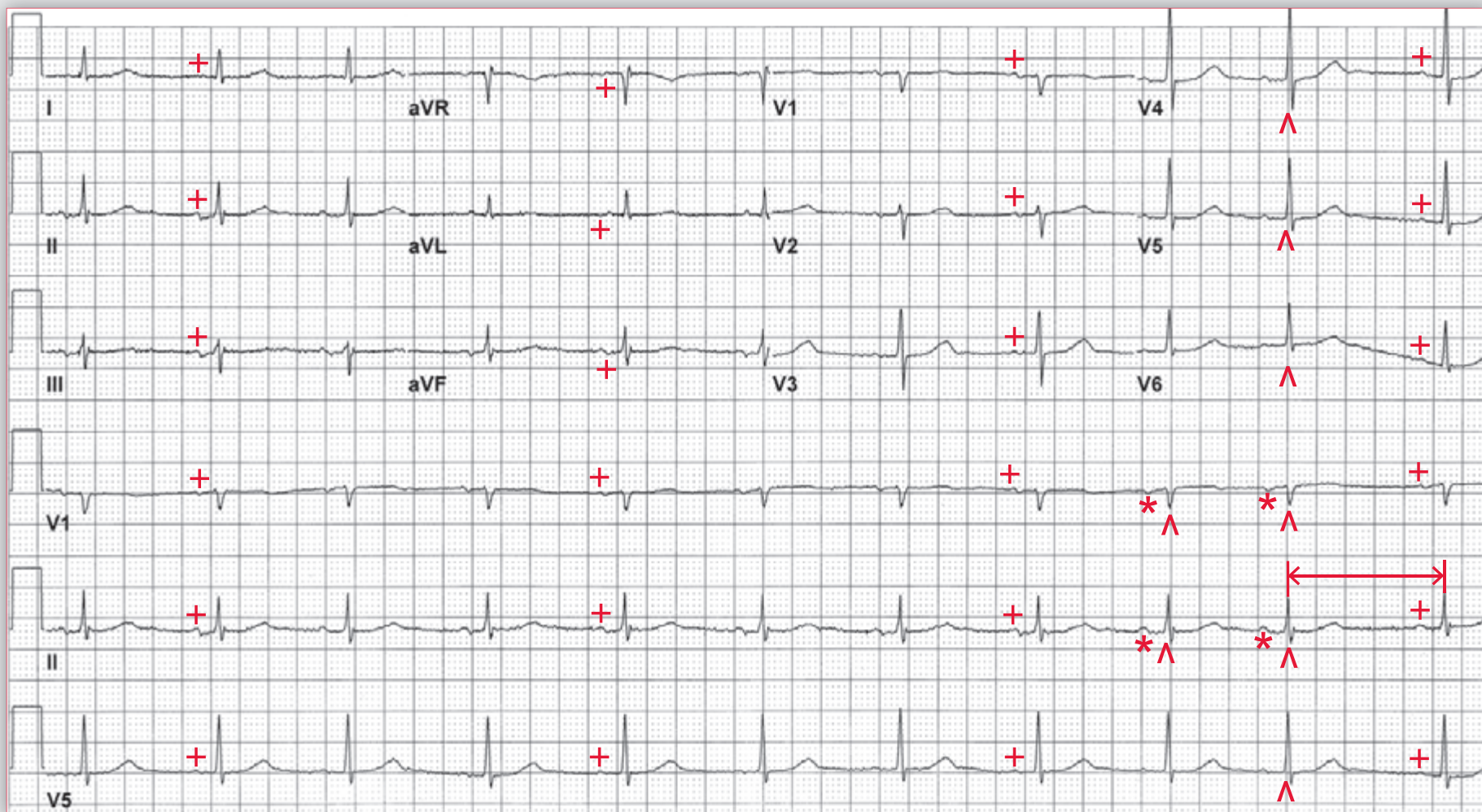
QRS and ST-T wave changes. In contrast, each QRS complex of a supraventricular tachycardia, regardless of where it originates (sinus node, atrial myocardium, or AV node) is always conducted to the ventricles via the same pathway (*ie*, His-Purkinje or accessory pathway) and hence the activation and repolarization sequence is always the same for each complex. Lastly, there are RS complexes in leads V4–V6 and the R wave is wider than the S wave, reflecting abnormal conduction of the initial portion of ventricular activation—another feature of a ventricular complex.

It should be noted that the small negative waveforms noted before the QRS complex in lead aVF are actually part of the QRS complex if the maximum QRS duration as noted in lead aVL is compared to the QRS complex in lead aVF (||).

The QRS morphology of the ventricular tachycardia resembles a right bundle branch block and has a leftward axis (borderline for a left anterior fascicular block). In addition the QRS complex is not extremely wide. These features are consistent with a fascicular tachycardia, *ie*, involving one of the fascicles of the left bundle. In this case, it would be a left posterior fascicular tachycardia, resulting in the QRS morphology and axis noted. A left posterior fascicular tachycardia is the most common type of fascicular tachycardia. A fascicular tachycardia, which is a type of idiopathic left ventricular tachycardia, is also called verapamil-responsive ventricular tachycardia or Belhassen tachycardia. It is commonly seen in younger patients without heart disease and it is not usually associated with a risk of sudden cardiac death, as there is often no structural heart disease present.

continues

Podrid's Real-World ECGs



ECG 20B Analysis: Normal sinus rhythm, premature atrial complexes

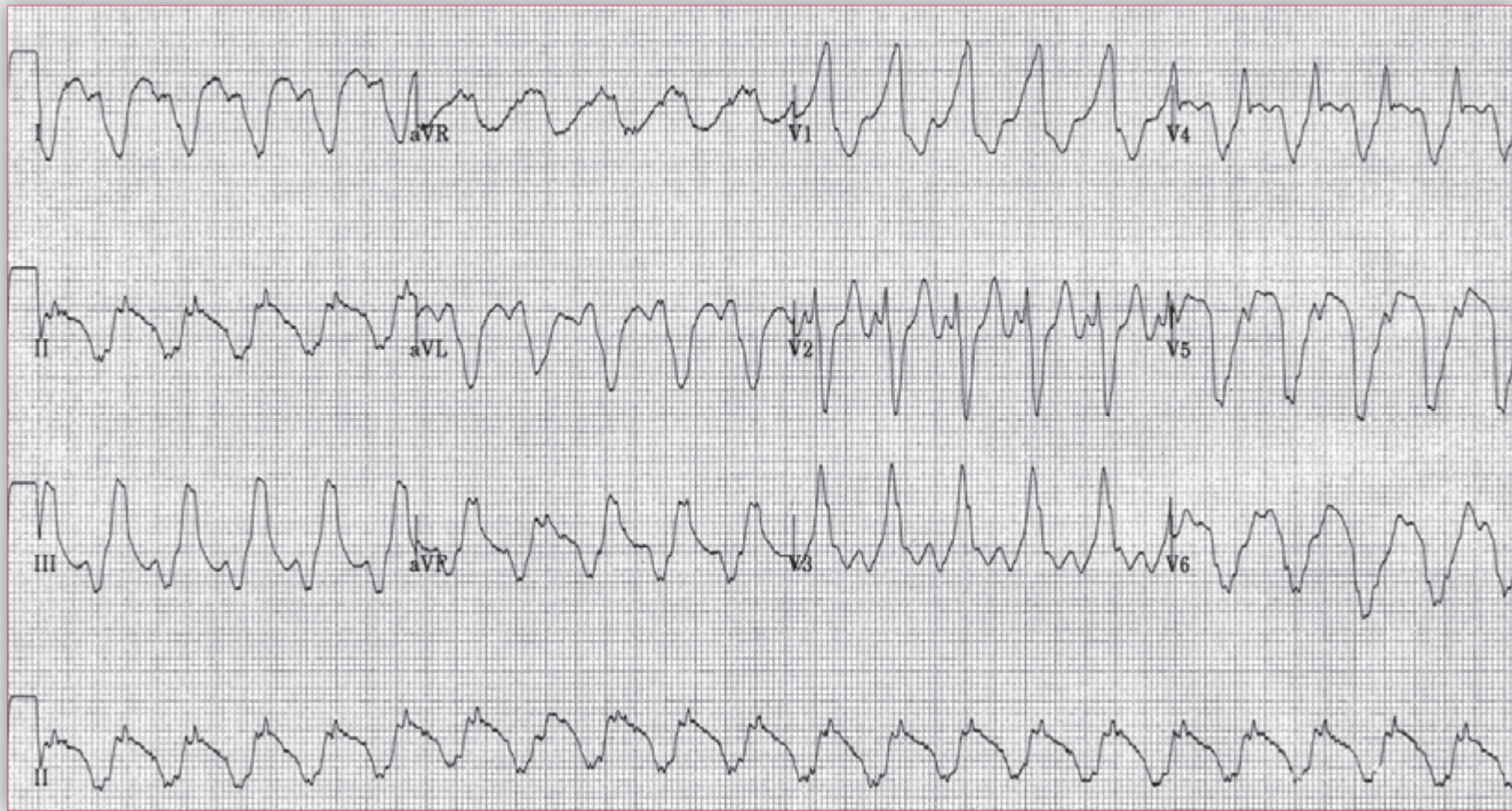
ECG 20B is from the same patient as ECG 20A. There is a regular rhythm at a rate of 64 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, V4–V6; hence this is a normal sinus rhythm. However, the tenth and eleventh QRS complexes are premature (^). There is a P wave before these complexes with a slightly different P-wave morphology (as noted in leads V1 and II) (*). These are premature atrial

complexes, after which there is a pause, or a longer RR interval (\leftrightarrow). The QRS complex duration is normal (0.08 sec) and there is a normal morphology and axis between 0° and $+90^\circ$ (positive QRS complex in leads II and aVF). The QT/QTc intervals are normal (400/410 msec). The QRS morphology and the PR interval of the captured beat in ECG 20A are the same as the morphology during normal sinus rhythm. ■

Core Case 21

A 78-year-old female with known ischemic cardiomyopathy presents to her local emergency department with acute-onset chest discomfort. She states she was woken from sleep with a “pressure” in her chest and shortness of breath. She activated emergency medical services.

ECG 21A

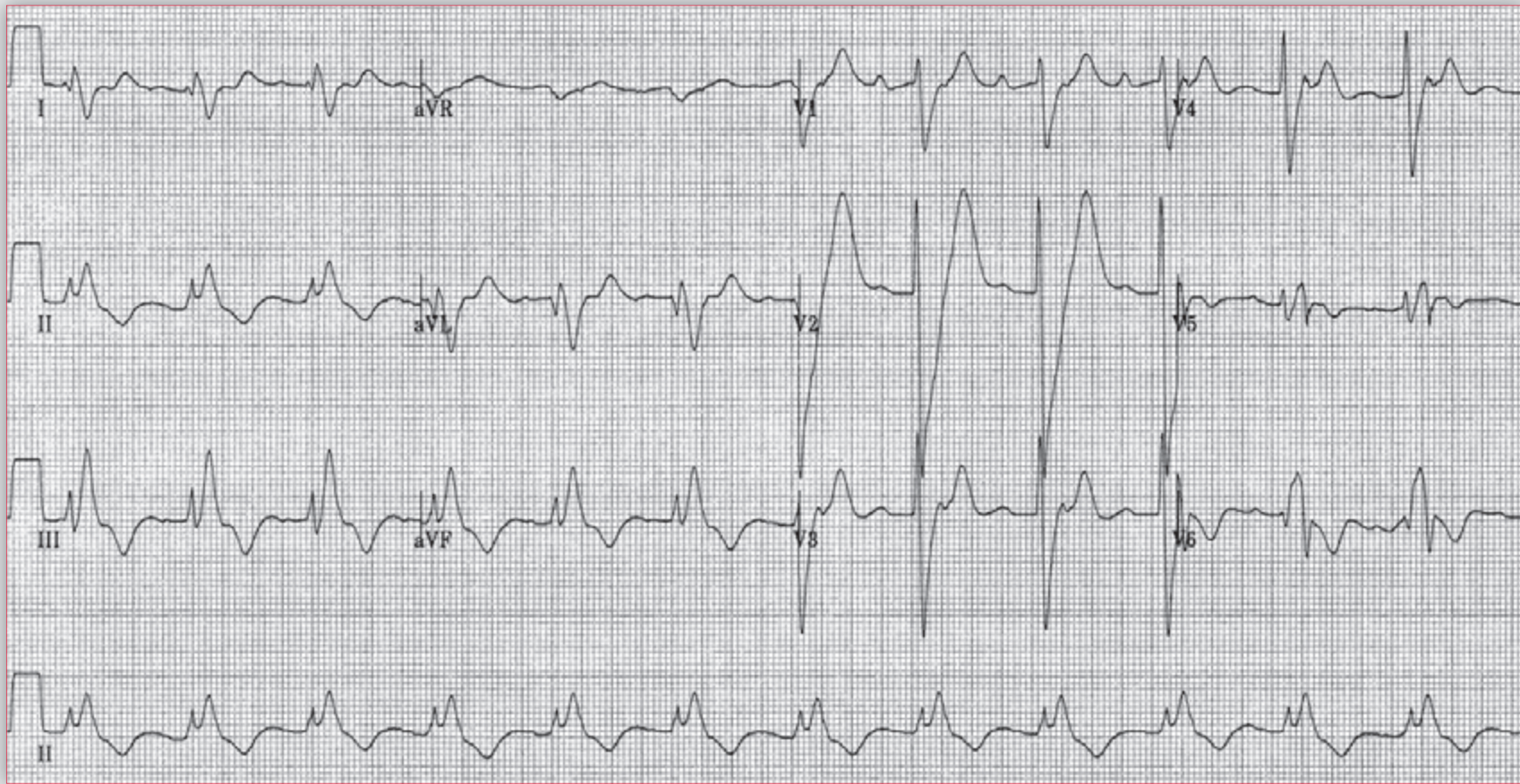


On arrival, she appears in moderate distress. Her vital signs are notable for a tachycardia and mild hypotension (100/70 mm Hg). The emergency medical technician hands you an ECG that was obtained en route (ECG 21A).

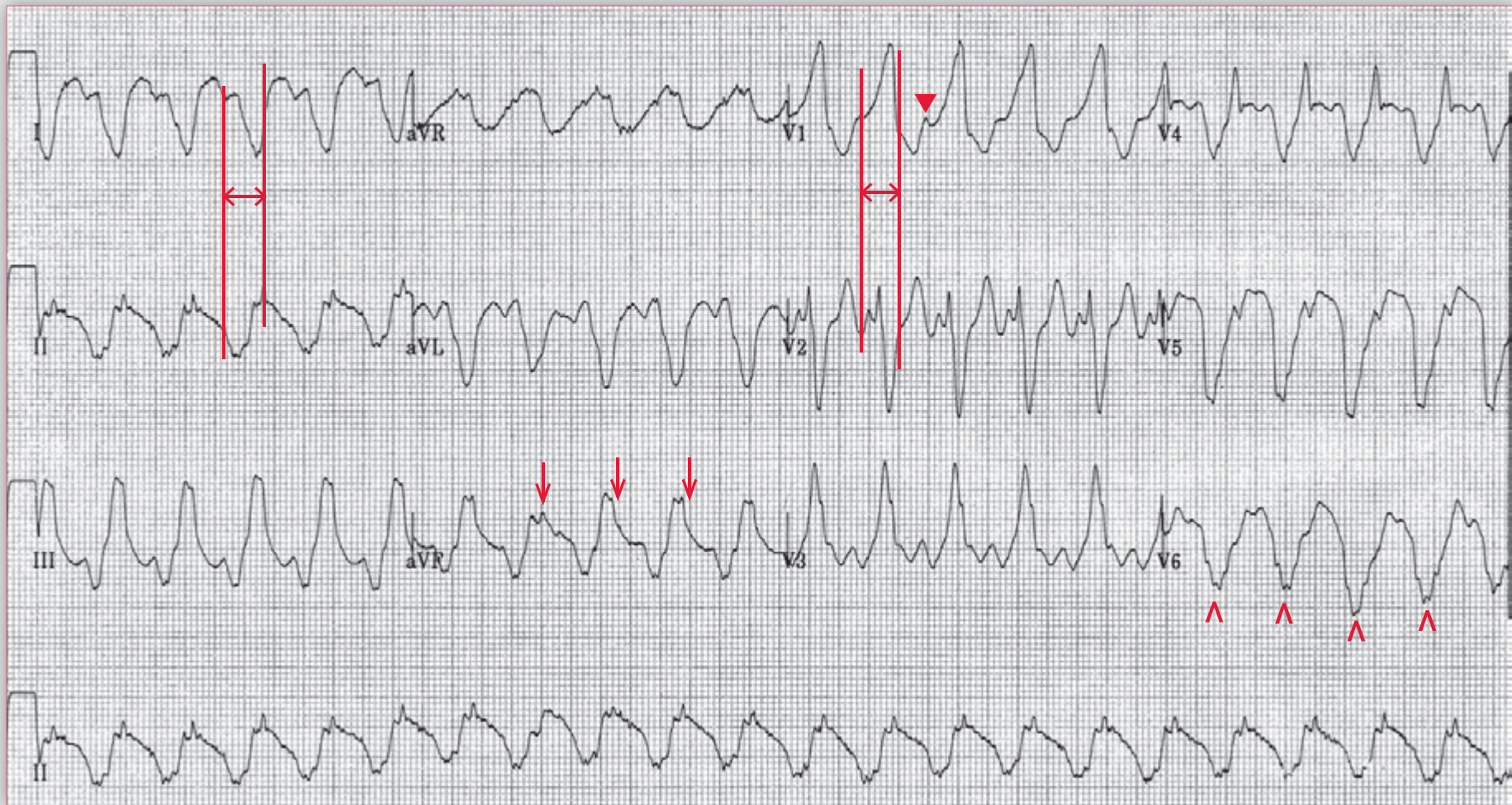
The emergency department nurse obtains a repeat ECG and hands this to you (ECG 21B).

Comparing the two tracings, what proximate cause for the patient's presentation is suggested?

ECG 21B



Podrid's Real-World ECGs



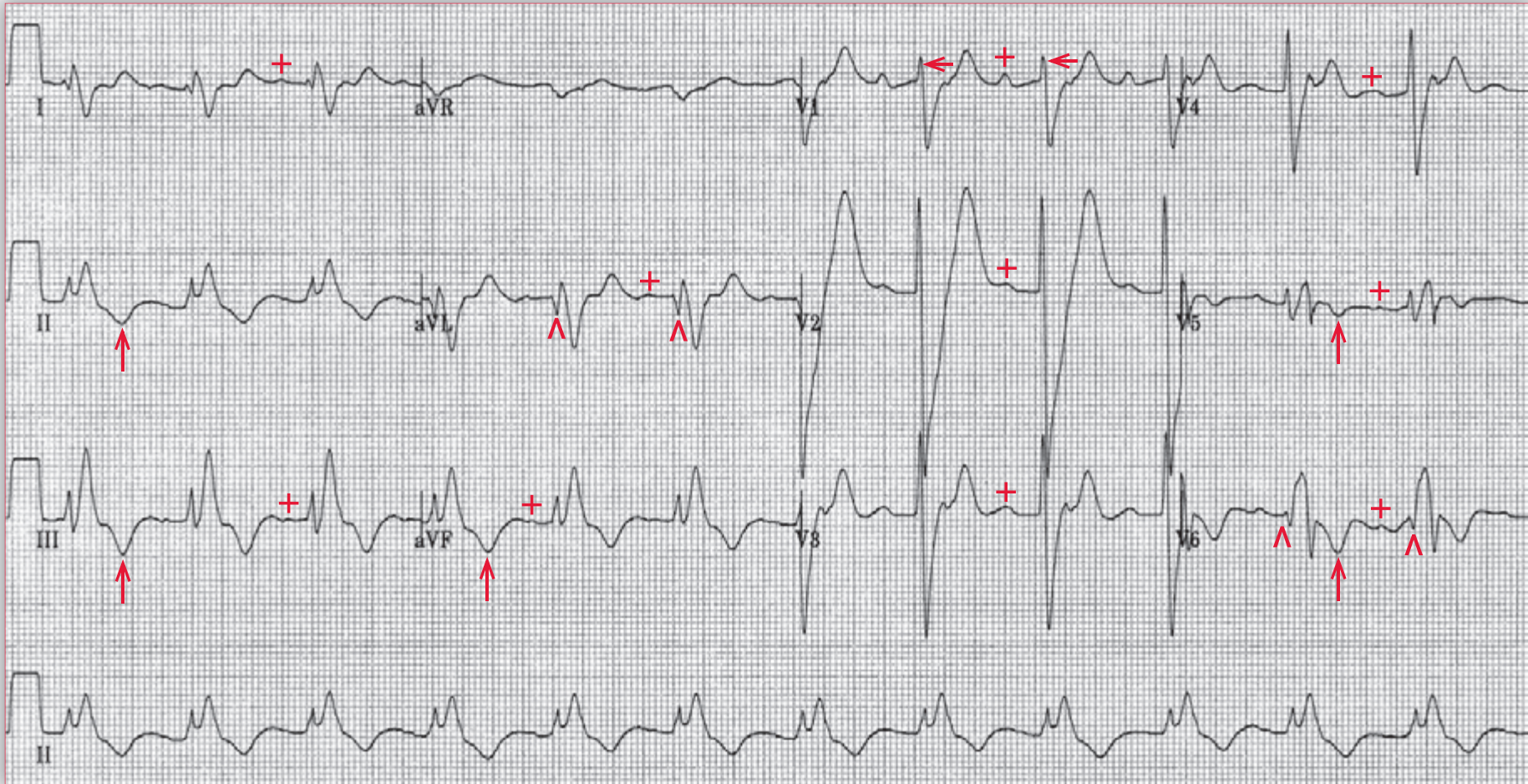
ECG 21A Analysis: Ventricular tachycardia (sustained monomorphic)

ECG 21A shows a regular rhythm at a rate of 130 bpm. The QRS complex duration is prolonged (0.22 sec) and the morphology is abnormal, resembling neither a typical right nor left bundle branch block. In addition, there is an indeterminate axis between $\pm 180^\circ$ and -90° (QRS complex negative in lead I and aVF). There are no constant P waves seen before or after the QRS complexes. It should be noted that the waveform before the QRS complex in leads I and V2 is part of the QRS complex and not a P wave. This is established by measuring the maximum QRS width as in lead V1 (\leftrightarrow) and comparing the duration to that seen in leads I and V2 (\parallel). There are occasional P waves seen, for example before the third complex in lead V1 (\blacktriangledown). The presence of a

P wave in front of some, but not every, QRS complex is diagnostic for AV dissociation. There are also subtle differences in QRS morphology (for example in lead V6) (\wedge) and ST-T waves (for example, lead aVF [\downarrow]). Hence this rhythm has a number of findings that are characteristic of ventricular tachycardia (*ie*, indeterminate axis, evidence of AV dissociation, changes in ST-T waves and QRS complex morphology). The width of the QRS complex (0.22 sec), which is wider than is usually seen in ventricular tachycardia, suggests that there is an underlying severe cardiomyopathy accounting for a significant degree of intraventricular conduction delay. It is also possible that hyperkalemia is present.

continues

Podrid's Real-World ECGs



ECG 21B Analysis: Normal sinus rhythm, intraventricular conduction delay, right axis deviation (left posterior fascicular block), first-degree AV block (prolonged AV conduction)

ECG 21B is from the same patient as ECG 21A and is the baseline ECG. There is a regular rhythm at a rate of 78 bpm. There are P waves seen before each QRS complex (+) with a stable PR interval (0.28 sec). The P waves are positive in leads I, aVF, and V4–V6. Hence this is a normal sinus rhythm with a first-degree AV block. The QRS complex duration is prolonged (0.20 sec). Although it has characteristics of a left bundle branch block, there is a significant septal R wave in lead V1 (←) and initial Q waves in leads aVL and V6 (^). Septal forces are not present with a left bundle branch block, as the septal innervation comes from a septal branch of the left bundle, with activation occurring from left to right (accounting for the R wave in V1 and the septal Q waves in leads I, aVL, and V4–V6). Hence this is an intraventricular conduction delay. The marked increase in QRS duration is characteristic of a severe dilated cardiomyopathy. The axis is rightward between +90° and +180° (negative QRS complex in lead I and positive in lead aVF). The presence of a rightward axis is another feature that excludes the

presence of a left bundle branch block. There are no left-to-right forces seen with a left bundle branch block and all forces are directed in a right-to-left direction. There are nonspecific T-wave abnormalities in leads II, III, aVF, and V5–V6 (↑). Noted are the marked changes in the QRS complex morphology in ECG 21A compared to the baseline QRS complex in this ECG, further supporting ventricular tachycardia as the etiology of the tachycardia in ECG 21A.

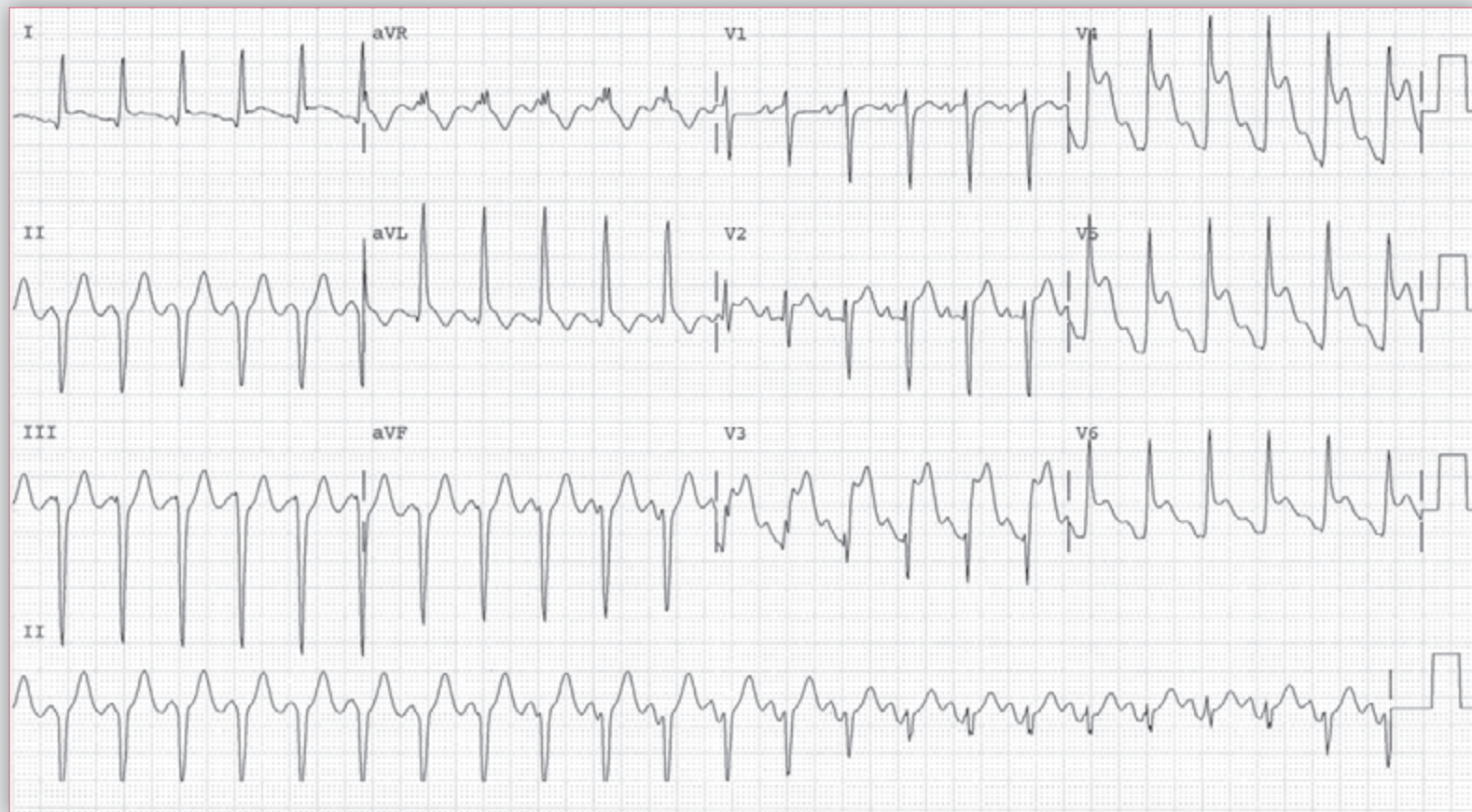
This patient with an underlying cardiomyopathy presents with ventricular tachycardia. Although the arrhythmia is not associated with significant hemodynamic compromise, the arrhythmia is associated with symptoms of angina and shortness of breath. The occurrence of ventricular tachycardia in a patient with underlying structural heart disease is an indication for the implantation of an automatic cardioverter-defibrillator. ■

Core Case 22

A 66-year-old female is admitted to the hospital with acute chest pain. An ECG is obtained (ECG 22A).

The patient is brought to the catheterization laboratory and a procedure is performed. Two hours later, a repeat ECG is obtained in the coronary care unit (ECG 22B).

ECG 22A



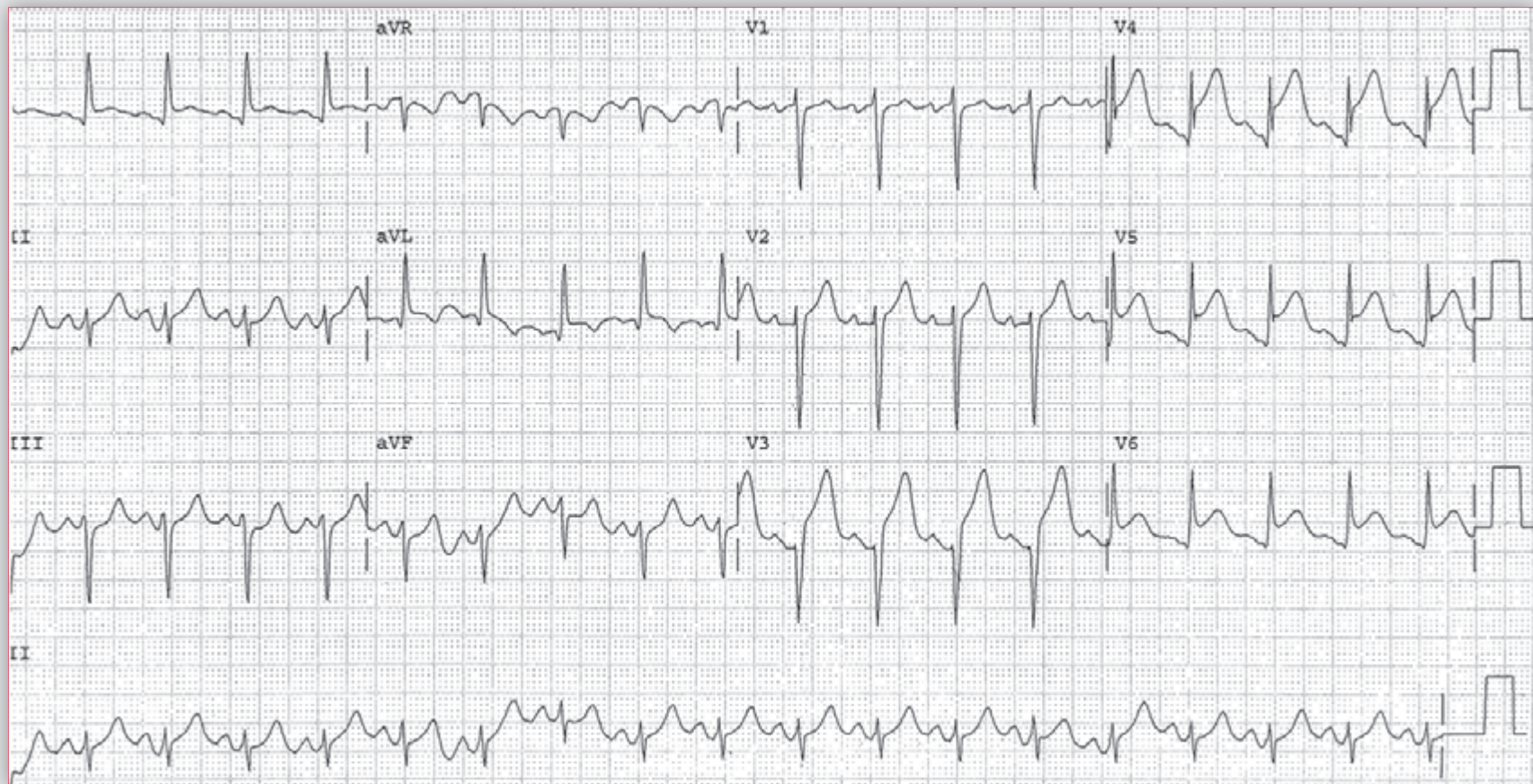
What abnormalities are noted on the tracing (ECG 22A)?

What clinical scenario is likely to be present given the arrhythmia depicted?

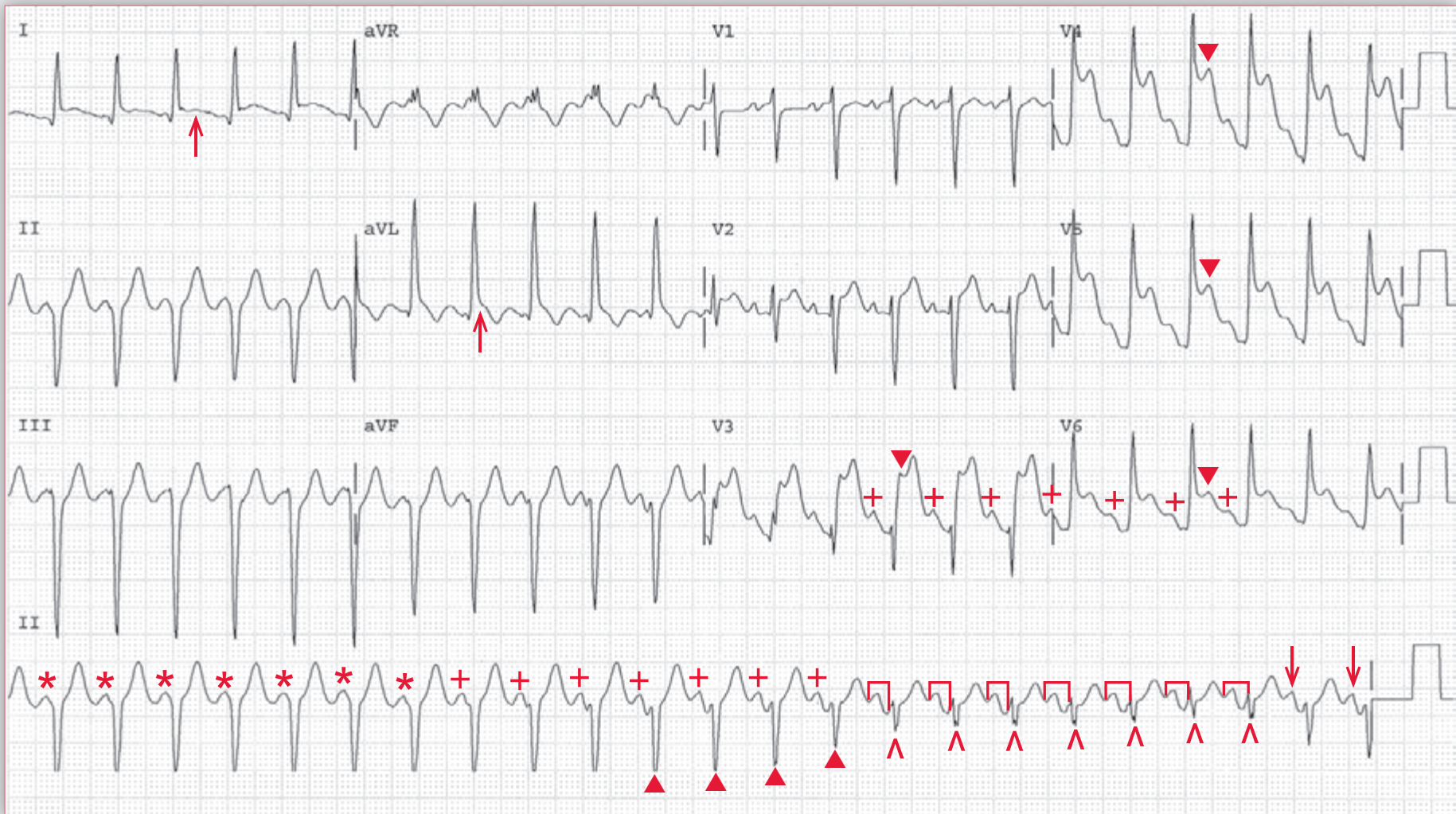
What coronary pathology is suggested?

What is your interpretation of the tracing (ECG 22B)?

ECG 22B



Podrid's Real-World ECGs



ECG 22A Analysis: Sinus tachycardia, acute anteroapical, anterolateral and lateral myocardial infarction, AV dissociation (isorhythmic dissociation), fusion complexes, ventricular tachycardia

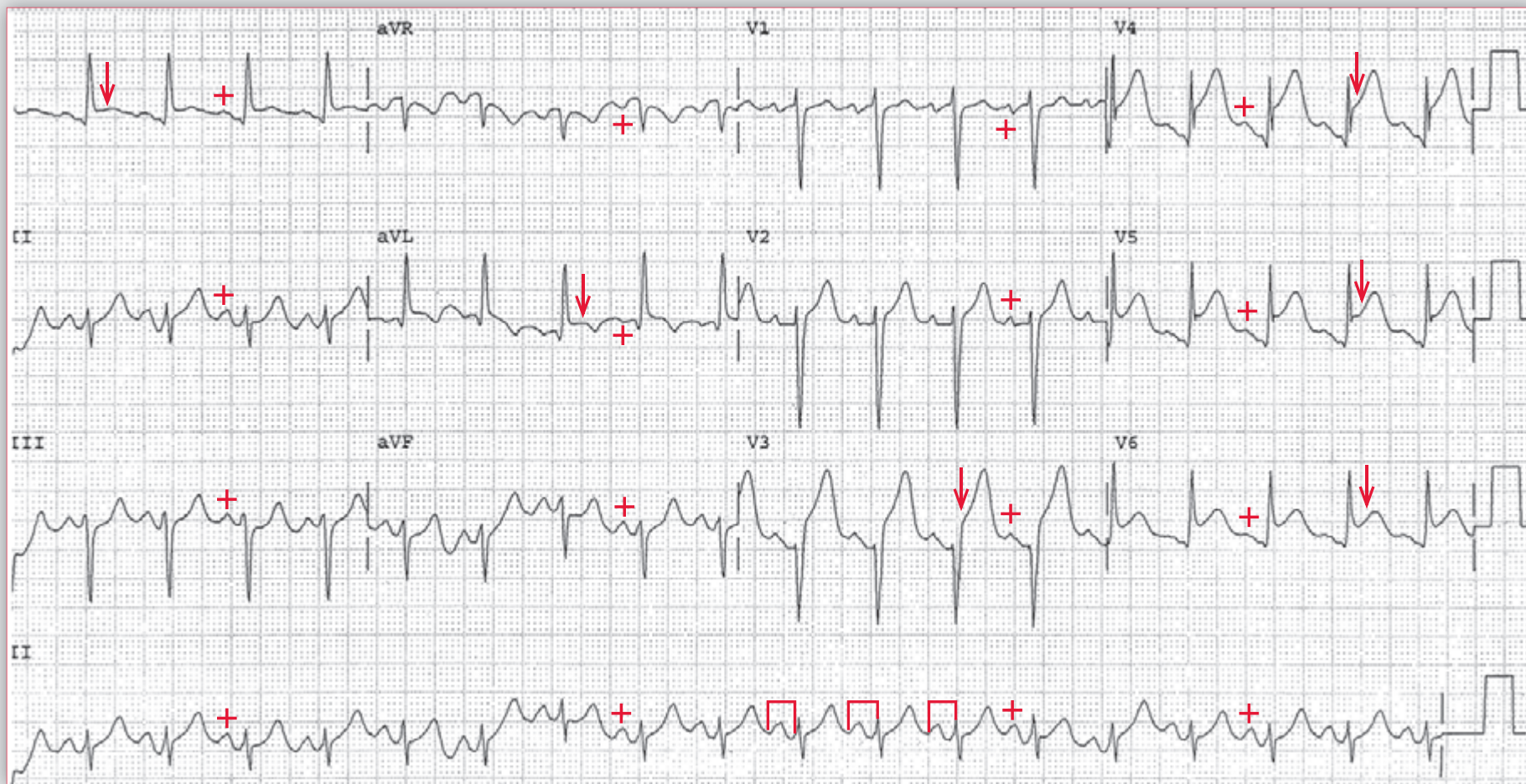
ECG 22A shows a regular rhythm at a rate of 140 bpm. In the initial portion of the ECG (first 14 QRS complexes), there are regular QRS complexes, each with a duration of 0.12 second. Although there are no P waves seen in the beginning of the ECG, P waves (+) become obvious by the eighth QRS complex in the rhythm strip. Indeed, there are P waves in the first 7 QRS complexes, which are superimposed at the beginning of the QRS complex (*). It should be noted that the PR interval gradually increases as the QRS complex duration decreases and changes in morphology. By the fifteenth through the twenty-first QRS complex, there is a stable PR interval (□) (0.16 sec) and a stable QRS morphology (^). The last two QRS complexes have a P wave before them (↓), but the PR is slightly shorter, the QRS complex morphology is slightly different, and the QRS complex duration is increasing. Hence there is evidence of AV dissociation present with a wide QRS complex. The changing morphology and duration of the QRS complex that occurs as the PR interval lengths suggests that these are fusion beats (especially complexes 11–14 [▲] where the QRS complex morphology is intermediate between the wide ones and the captured complexes), while

the fifteenth through the twenty-first QRS complexes are captured. At the end, there is again fusion (last two QRS complexes). Hence this is a sinus tachycardia with AV dissociation and a ventricular tachycardia. Since the ventricular rate and atrial rate are identical, this is termed isorhythmic dissociation. It is not clear if this is complete heart block with an escape ventricular rhythm or an accelerated idioventricular rhythm, *ie*, ventricular tachycardia. The occurrence of intermittent fusion and capture means that this is an accelerated ventricular rhythm, *ie*, ventricular tachycardia. When there is a subtle increase in the sinus rate (or a subtle decrease in the ventricular rate), AV capture occurs.

In addition, there is pronounced ST-segment elevation in leads V3–V6 (▼). The ST segments still have a concave morphology. Given the clinical history, the ECG changes are consistent with an acute anterolateral myocardial infarction. Although the complexes in leads I through aVF are ventricular, there is also ST-segment elevation in leads I and aVL (↑), suggesting that there is also involvement of the lateral wall.

continues

Podrid's Real-World ECGs



ECG 22B Analysis: Sinus tachycardia, left anterior fascicular block, acute anteroapical, anterolateral and lateral myocardial infarction

ECG 22B is from the same patient as ECG 22A and was obtained 2 hours later. There is a regular rhythm at a rate of 110 bpm. A P wave (+) is present before each QRS complex, and there is a constant PR interval (0.16 sec) (\square), which is the same as seen with the captured QRS complexes in ECG 22A. The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. The QRS complexes have the same width and morphology as the captured complexes in ECG 22A. There is an extreme left axis between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF with an rS morphology); this is a left anterior fascicular block. The QT/QTc intervals are normal (330/410 msec). There is significant ST-segment elevation (\downarrow) in leads V3–V6 and ST elevation also in leads I and aVL. The ST segments are convex, merging with the T waves. The T waves are symmetric. Hence these are typical features of an acute myocardial infarction of the anteroapical, anterolateral and lateral walls.

The ventricular rhythm is termed an accelerated idioventricular tachycardia when the ventricular rate is 60 to 100 bpm or ventricular tachycardia when the rate is > 100 bpm. Although the ventricular rate is 140 bpm (and hence considered a ventricular tachycardia), given the fact that the sinus rate is also 140 bpm, this might actually be considered an accelerated idioventricular rhythm, which is typically seen in instances of coronary reperfusion. In this case, as the patient's presenting ECG showed this rhythm, it is likely that there was spontaneous re-canalization of the infarct artery. Spontaneous thrombolysis or reperfusion of a coronary artery in the context of acute myocardial infarction is estimated to occur in approximately 20% of presenting cases and is associated with improved clinical outcomes.

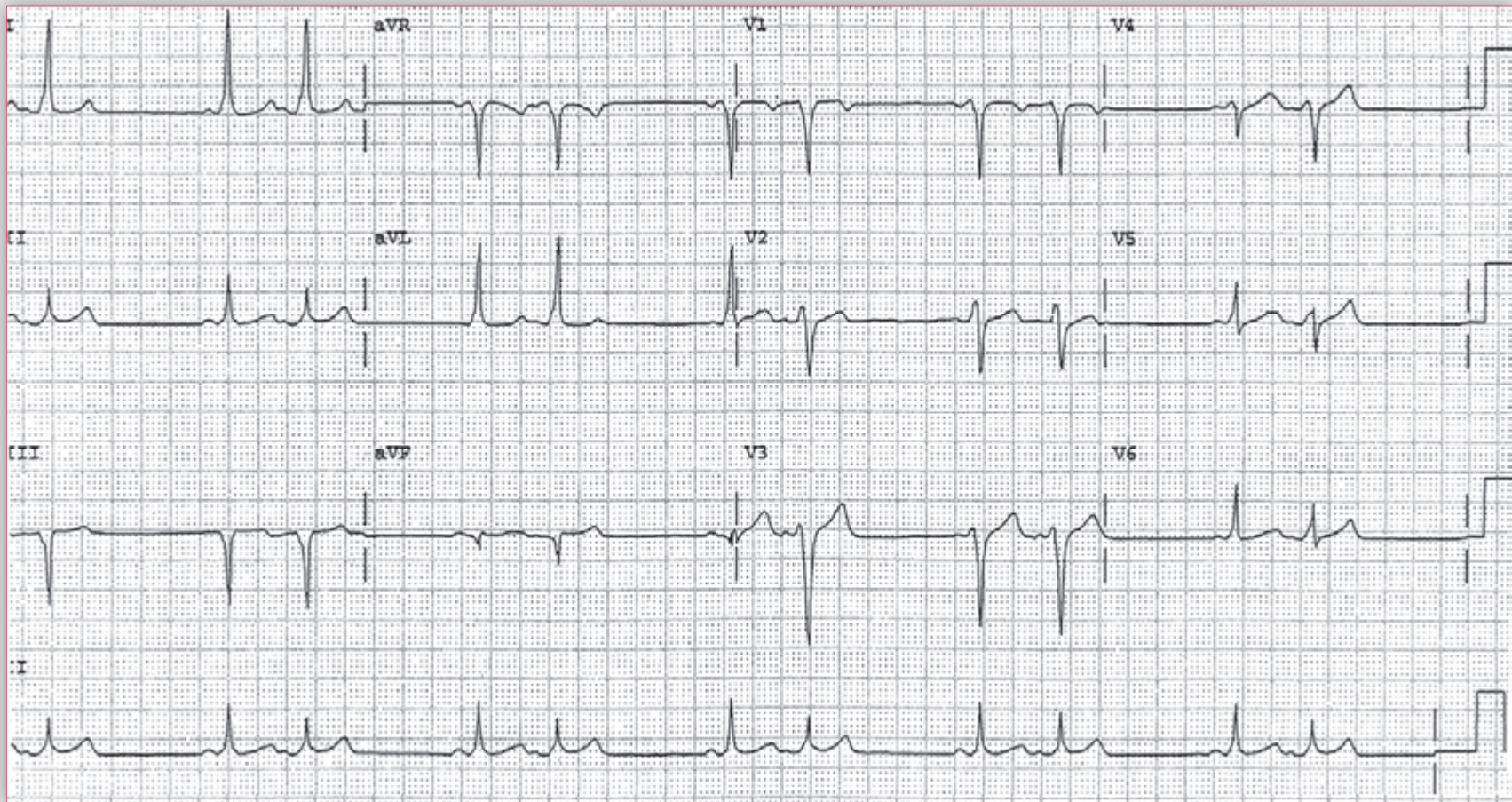
The ECG changes associated with an acute myocardial infarction are first hyperacute T waves, followed by ST-segment elevation. Initially the ST segments maintain their normal concave morphology, but then change to become convex, merging with the T waves. This change in pattern is seen between ECGs 22A to 22B. ■

Notes

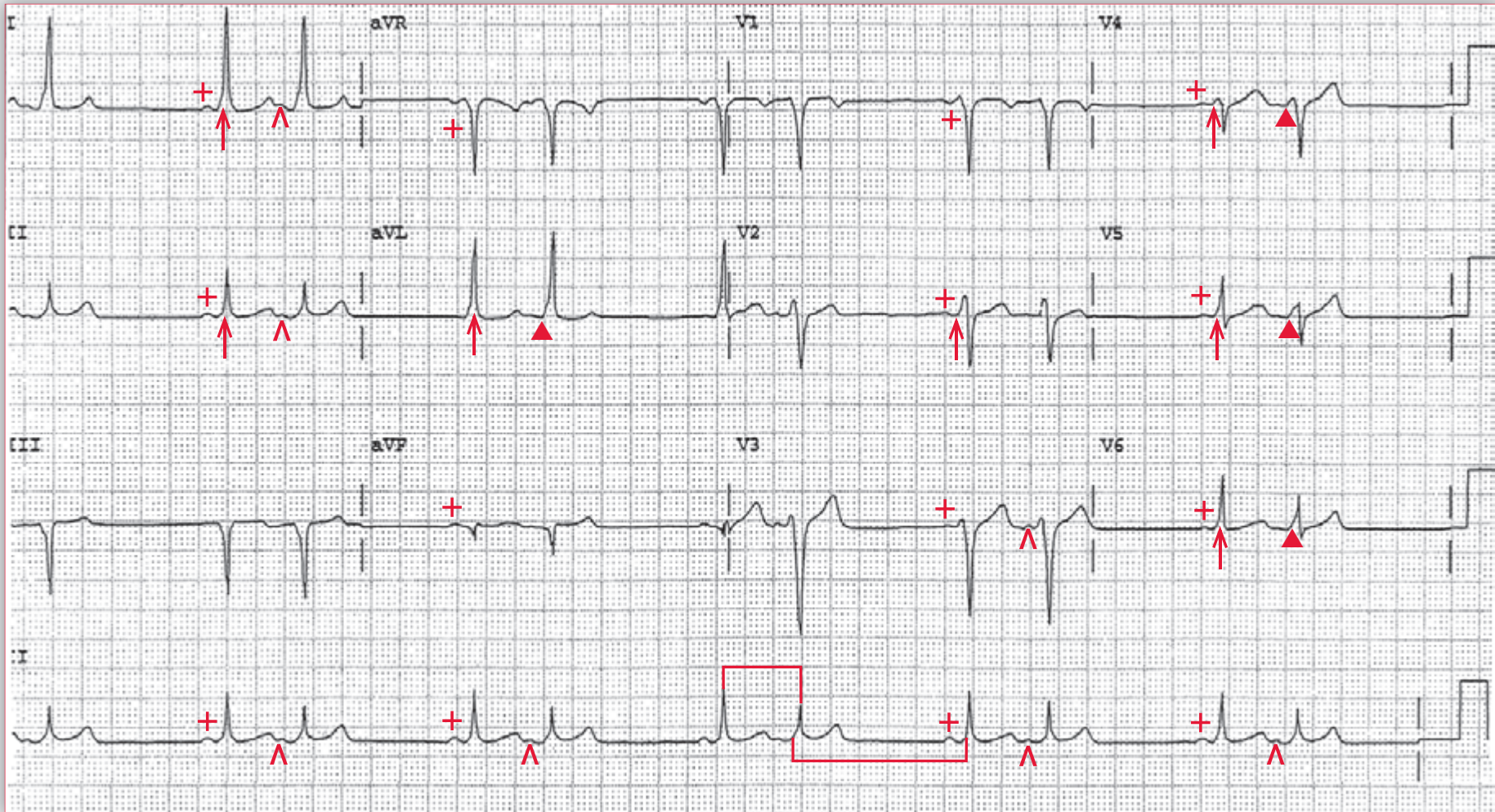
A 24-year-old man presents to his primary physician with complaints of intermittent palpitations. He has noted these symptoms on and off for a year. They occur and remit spontaneously and can last for minutes up to an hour at a time. He denies syncope. He is otherwise healthy and a general review of symptoms is unremarkable.

The physician obtains an ECG.

What abnormalities are noted and what clinical syndrome is suggested?



Podrid's Real-World ECGs



ECG 23 Analysis: Normal sinus rhythm, Wolff-Parkinson-White pattern, premature atrial complexes with increased preexcitation atrial bigeminy

There is a regularly irregular rhythm with an average rate of 78 bpm. There appears to be group beating with long (⏏) and short (⏏) RR intervals. After each long RR interval, there is a QRS complex that is preceded by a P wave (+) with a PR interval that is short, but constant (0.10 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus rhythm with a short PR interval. The QRS complex duration is prolonged (0.12 sec), and there is a slurred upstroke to the QRS complex (†) that is a delta wave. The delta wave causes the base of the QRS complex to be widened, while the rest of the QRS complex is narrow. Hence this is a sinus complex with a Wolff-Parkinson-White (WPW) pattern. The QT/QTc intervals are 400/410 msec.

Following this sinus complex, there is an early or premature P wave (^) that is different in morphology from the sinus P wave. This is a premature atrial complex. As every other complex is an atrial premature complex, this is termed atrial bigeminy. The PR interval of each of these premature complexes is short and the same as the sinus complexes (0.10 sec) and the QRS complex has a delta wave (▲). However, the QRS complex duration is slightly longer than the sinus complex (0.14 sec) and the delta wave is more prominent, most obviously in leads V4–V6. The WPW complex represents fusion between conduction via the normal AV node–His-Purkinje system and the accessory pathway. The PR interval and delta wave (and hence QRS complex width)

is related to a balance of conduction between these two pathways. The wider QRS complex and more prominent delta wave indicates that more of ventricular activation of the premature atrial complex is originating via the accessory pathway compared to the sinus complex. This is based on the fact that the premature atrial stimulus occurs at a shortened PP interval and reaches the AV node earlier, before it has completely repolarized. Hence AV conduction velocity is decreased; this is termed decremental conduction, *ie*, at faster rates, the rate of conduction through the AV node decreases. While the conduction through the AV node is slower, there is no change in the conduction velocity via the accessory pathway. Hence more of the ventricle is activated via the accessory pathway rather than the normal His-Purkinje system, resulting in a broader delta wave and wider QRS complex. As initial ventricular activation is through the accessory pathway and the conduction velocity through this pathway is constant, the PR intervals of the sinus complex and premature atrial complex are the same. Another possible cause for the wider QRS complex of the premature atrial complex is that the ectopic atrial focus is closer to the accessory pathway.

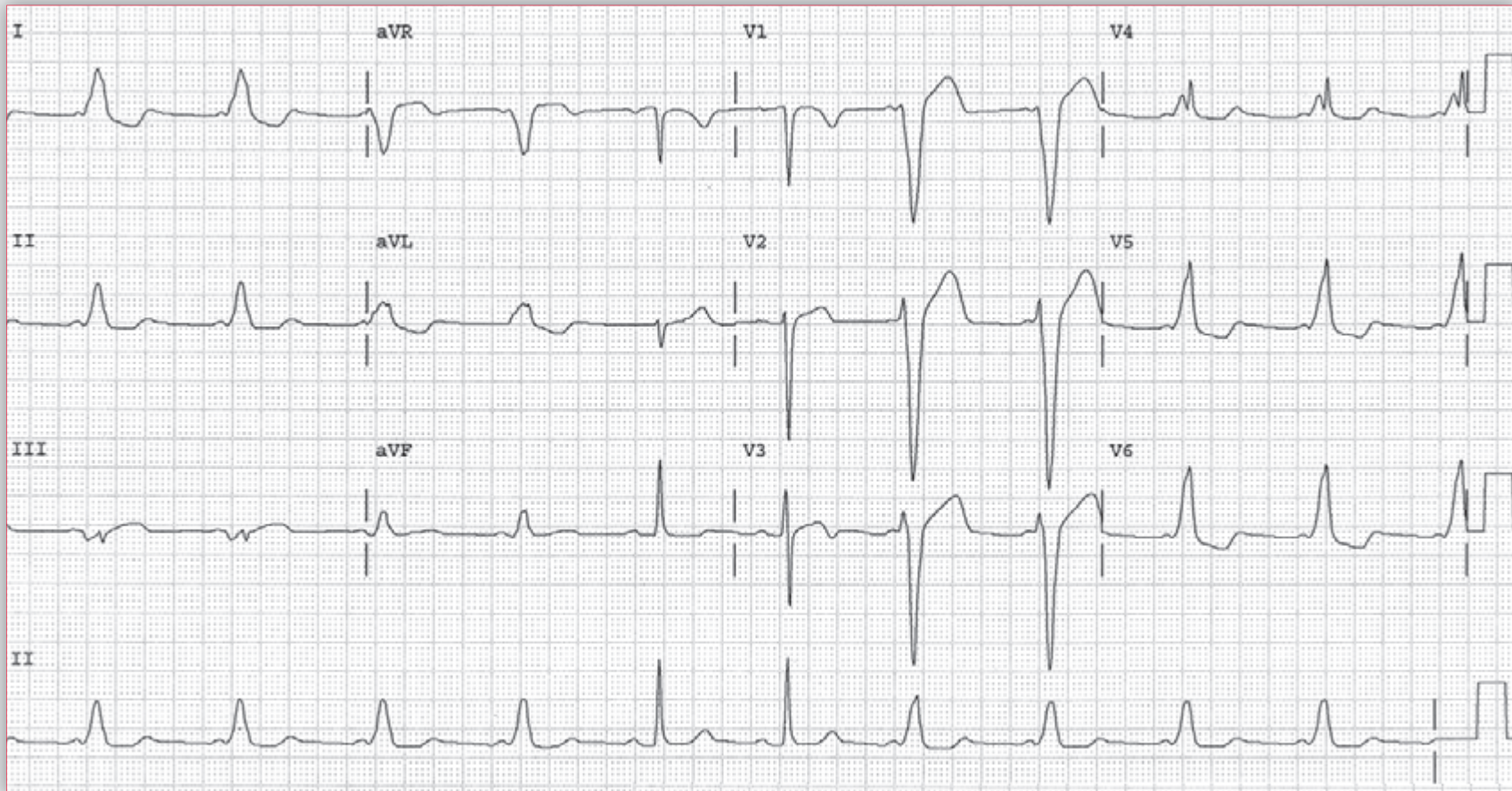
The ECG demonstrates QRS complexes that have a WPW pattern. The WPW syndrome represents the arrhythmias that are associated with WPW. The WPW pattern is identified by the electrocardiographic pattern of a short PR interval and a delta wave. ■

Notes

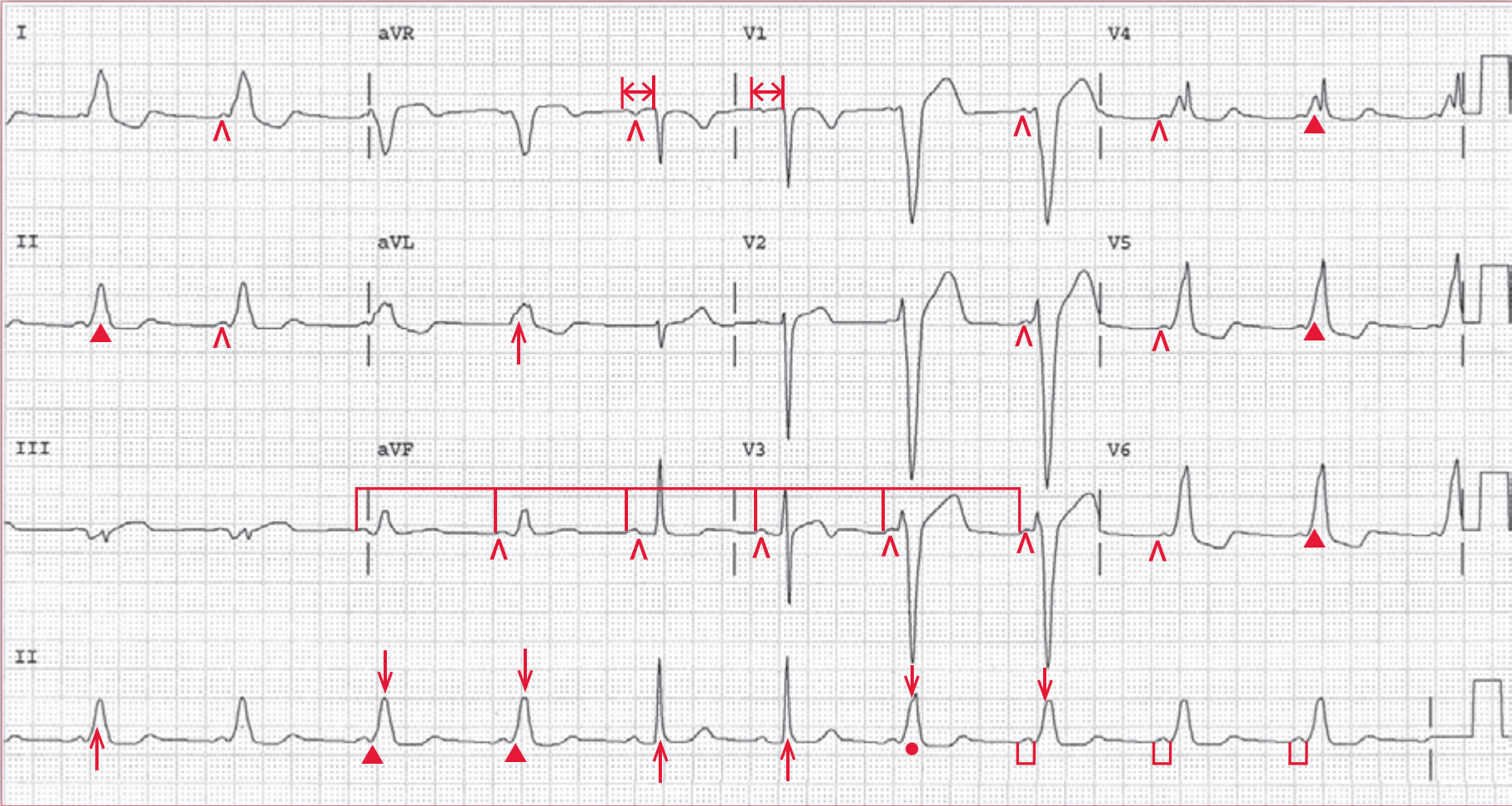
A 16-year-old healthy female with a strong family history of sudden cardiac death undergoes a screening ECG.

What abnormalities are noted?

What is the mechanism of sudden death associated with the abnormality seen?



Podrid's Real-World ECGs



ECG 24 Analysis: Normal sinus rhythm, Wolff-Parkinson-White pattern (intermittent)

There is a regular rhythm at a rate of 64 bpm. The QRS complexes have two different durations (narrower [↑] at 0.10 sec and wider [↓] at 0.18 sec). The axis is normal between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (420/430 msec). There is a P wave (^) before each QRS complex, and the PP intervals are constant (□). The P wave is positive in leads I, II, aVF, and V4–V6. Hence there is an underlying normal sinus rhythm, but the PR intervals are not constant. The PR interval before the narrow QRS complexes is stable (0.20 sec) (↔), while the PR interval before the wide QRS complexes are also stable, but shorter (0.10 sec) (⊐). The narrow QRS complexes are only seen in a few leads, but they appear to have a normal morphology. The wide QRS complexes have a slurred upstroke or delta wave (▲), and along with the short PR interval, these have a Wolff-Parkinson-White (WPW) pattern. Therefore, this ECG shows intermittent WPW.

The WPW pattern represents fusion between conduction via the normal AV node–His-Purkinje system and conduction via an accessory pathway, known as a bundle of Kent, which is a connection between the atria and ventricles. The width of the QRS complex or the extent of the delta wave is determined by conduction via the AV node. Conduction through the accessory pathway is fixed and is all or none. However,

whether conduction will occur via the accessory pathway is determined by its refractoriness. If it has a short refractory period, there is likely to be conduction via the accessory pathway at all heart rates. If the refractoriness is relatively long, then conduction via this pathway may not occur when the heart rate increases. In this situation, preexcited QRS complexes will be intermittently present. Indeed, the presence of intermittent WPW pattern, as is seen in this ECG, generally indicates that the refractoriness of the accessory pathway is relatively long, and hence rapid conduction via this pathway is not likely to occur. In this situation, there is a decreased risk for rapid ventricular rates associated with rapid atrial rates, particularly during atrial fibrillation. It is the rapid ventricular response rate that may be 350 bpm faster and can precipitate ventricular fibrillation and hence sudden cardiac death, even in a structurally normal heart.

Also determining the presence and extent of preexcitation is conduction through the AV node. If AV nodal conduction is slow, more of the ventricular myocardium is activated via the accessory pathway and hence the delta wave is more prominent and the PR interval shorter. If AV nodal conduction is fast, then less of the ventricular myocardium is activated via the accessory pathway, and the delta wave is less prominent and the PR interval is longer. ■

Notes

An 18-year-old male experiences a sudden episode of severe lightheadedness and near syncope while playing recreational soccer. The episode lasts about 3 minutes and then the symptoms suddenly resolve.

He is brought to the local emergency department. He denies any previous cardiac or medical history, and he has been healthy without medical diagnoses. He does not take regular medications, and he denies any drug or alcohol abuse.

In triage, he is noted to have a resting tachycardia and an ECG is obtained.

What is your interpretation of this ECG?

What are the possible causes for his presyncopal episode?



Podrid's Real-World ECGs



ECG 25 Analysis: Normal sinus rhythm, left atrial hypertrophy or abnormality, intermittent Wolff-Parkinson-White pattern, pseudo inferior wall myocardial infarction

There is a regular rhythm at a rate of 120 bpm. The QRS complexes have two different morphologies and durations. The narrow QRS complex (duration = 0.08 sec) and there is a normal morphology and axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (320/450 msec). The wide QRS complex (duration = 0.14 sec) has an abnormal morphology. There is a slurred upstroke noted (\uparrow), accounting for the increased QRS complex duration. This is a delta wave. The axis is extremely leftward axis between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF). The negative complex in leads II and aVF is due to a deep Q wave (\wedge), which is also seen in lead III (\wedge). This is indicative of an inferior wall myocardial infarction.

There is a P wave (+) seen before each QRS complex, and the PP interval is constant (\sqcup). The P wave is positive in leads I, II, aVF, and V4–V6. The P waves are prominently notched in leads V2–V4 (often referred to as a P-mitrale pattern), suggesting left atrial abnormality (hypertrophy). The PR intervals are different. The PR interval associated with the narrow QRS complex is 0.16 second while the PR interval associated with the wide QRS complex is short (0.08 sec). The short PR interval associated with the wide QRS complex that has a delta wave represents Wolff-Parkinson-White (WPW) pattern. The presence of inferior Q waves, only seen with the widened QRS complexes, is termed a pseudo infarction pattern indicating a posteroseptal bypass tract. As there is a positive delta wave in lead V1, the accessory pathway is left sided. As WPW is associated with direct myocardial activation via the accessory pathway, abnormalities affecting the ventricles

cannot be reliably diagnosed; hence this is a pseudo inferior wall myocardial infarction.

The alternating narrow and preexcited QRS complexes is termed intermittent WPW. In general, this indicates that the refractory period of the accessory pathway is relatively long and is not likely to conduct all the impulses when they occur at a rapid rate.

The occurrence of arrhythmias in patients with WPW constitutes a WPW syndrome. Patients with WPW have the anatomic substrate for the development of a reentrant arrhythmia known as atrioventricular reentrant tachycardia (AVRT). This is the result of a circuit formed by the two pathways that link the atria and ventricles, *ie*, the normal AV node–His–Purkinje system and the accessory pathway. These two tracts are linked by the atrial myocardium and the ventricular myocardium, forming a macro-reentrant circuit. As the two pathways have different electrophysiologic properties, there is the potential for the occurrence of a reentrant arrhythmia. It is generally provoked by a premature complex (ventricular or atrial), which is conducted via one pathway (antegradely or retrogradely) only, and then enters and conducts via the other pathway in the opposite direction. If this process continues, an arrhythmia occurs. Unless there is underlying structural or functional ventricular dysfunction, this arrhythmia generally does not cause syncope, especially in young, otherwise healthy patients with normal LV function. In addition, there are other arrhythmias seen in WPW that are generated in the atrial myocardium, but use the accessory pathway to conduct to the ventricles. These include atrial tachycardia,

continues

Notes

atrial flutter and atrial fibrillation. In this situation the ventricular rate is determined by the atrial rate as well as the ability of the accessory pathway to conduct. This is of particular concern when the atrial arrhythmia is atrial fibrillation as in this situation the atrial rate may be > 350 or often > 450 bpm. If the accessory pathway has a short refractory period and is therefore capable of being stimulated at a rapid rate, the ventricular response rate may be at a level above 350 bpm, which may be associated with near syncope or syncope. In addition, at this rapid rate ventricular fibrillation may be provoked, even in a structurally normal heart, resulting in sudden death—the most feared complication of WPW.

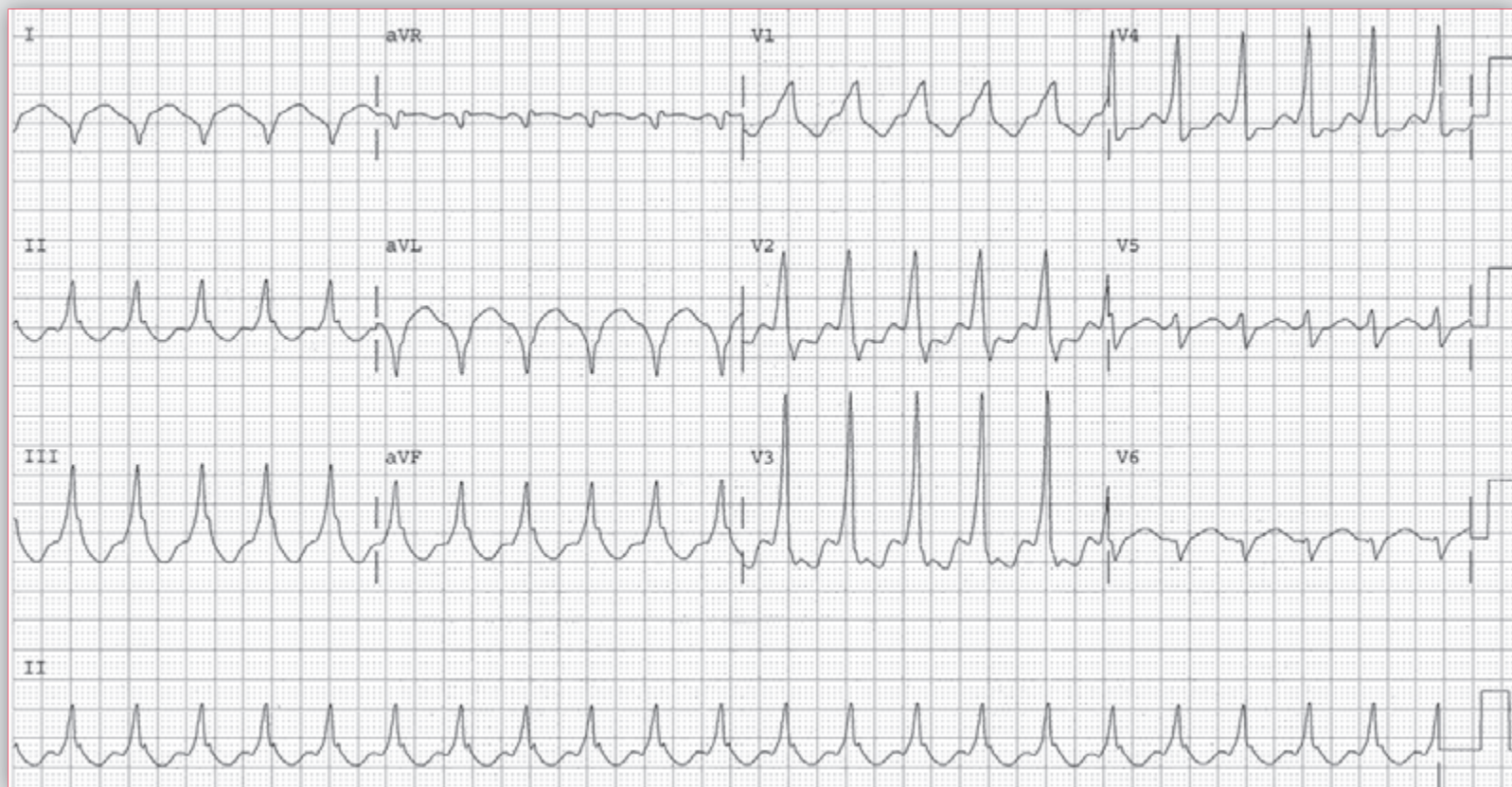
In the case presented, the suggestion that the accessory pathway has a long refractory period means that it is likely unable to be stimulated by atrial impulses at a rapid rate; hence this patient is at a low risk for sudden death. However, there is a possibility that an associated arrhythmia might be rapid enough to result in symptoms of dizziness and near syncope. ■

Core Case 26

A 32-year-old female presents to her primary physician with complaints of frequent episodes of anxiety and palpitations. She has had these episodes on and off for 10 years. She currently takes short-acting benzodiazepines as abortive therapy for anxiety, though she states that recently they don't seem to be very effective.

She describes these episodes in more detail as acute in onset, characterized by general feelings of anxiousness, associated with the feeling that her “heart is coming out of (her) chest,” and most recently mild dyspnea. The episodes seem to occur without warning, but are more frequent in the mornings. They similarly end

ECG 26A



without warning, but she has noticed that occasionally coughing can stop the episodes.

Her medical history is otherwise notable for anorexia nervosa. She does not take any medications other than the anxiolytics. Her family history is notable for a father with a “thick heart.”

Her exam is unremarkable. During the visit, she suddenly becomes very anxious and states she is having a “panic attack.” The physician feels her pulse and notices a tachycardia. An ECG is obtained (ECG 26A).

The episode is terminated by coughing and a post-tachycardia ECG is obtained (ECG B).

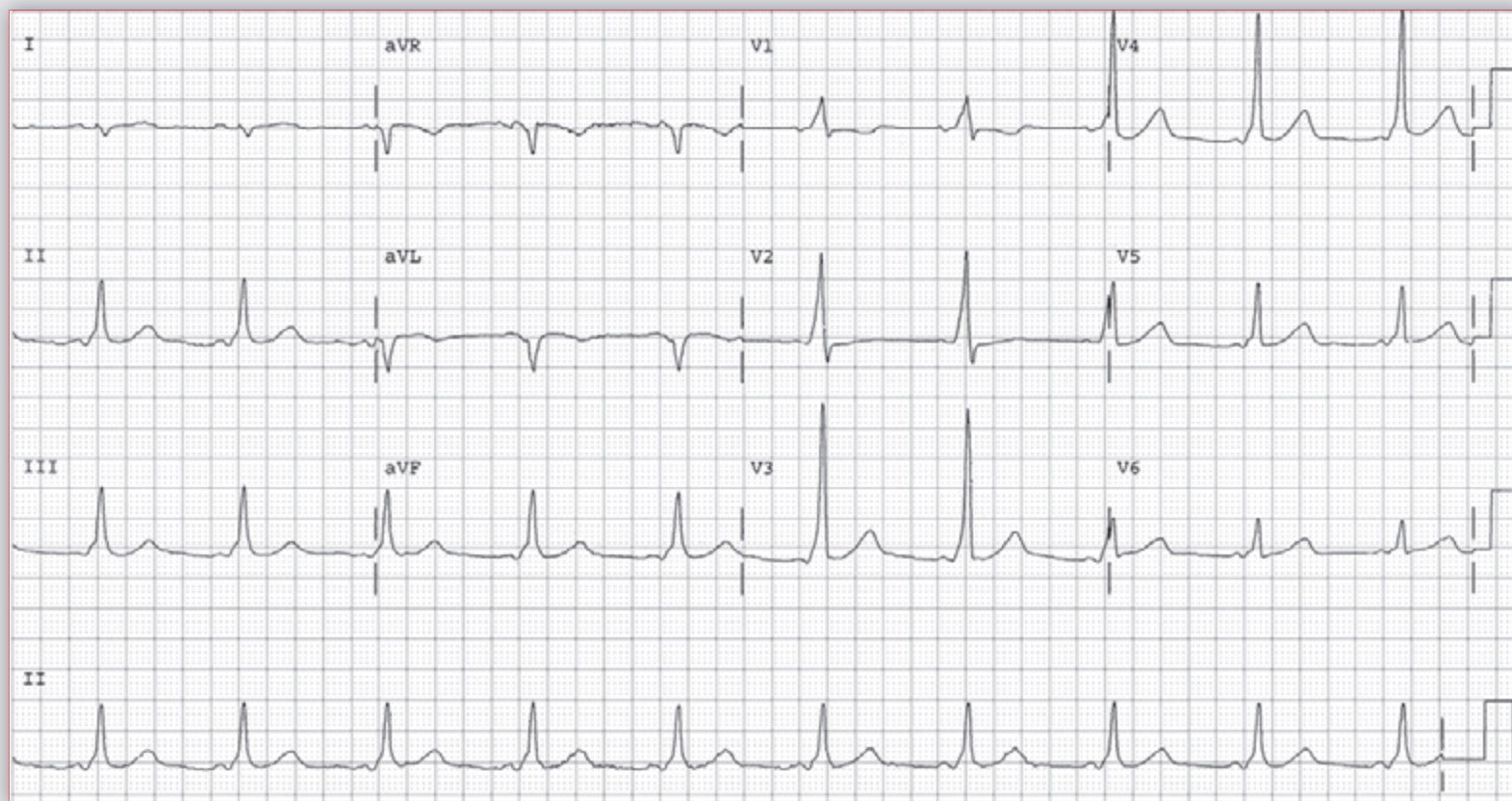
What is your interpretation of the tracing (ECG 26A)?

What diagnoses are suggested?

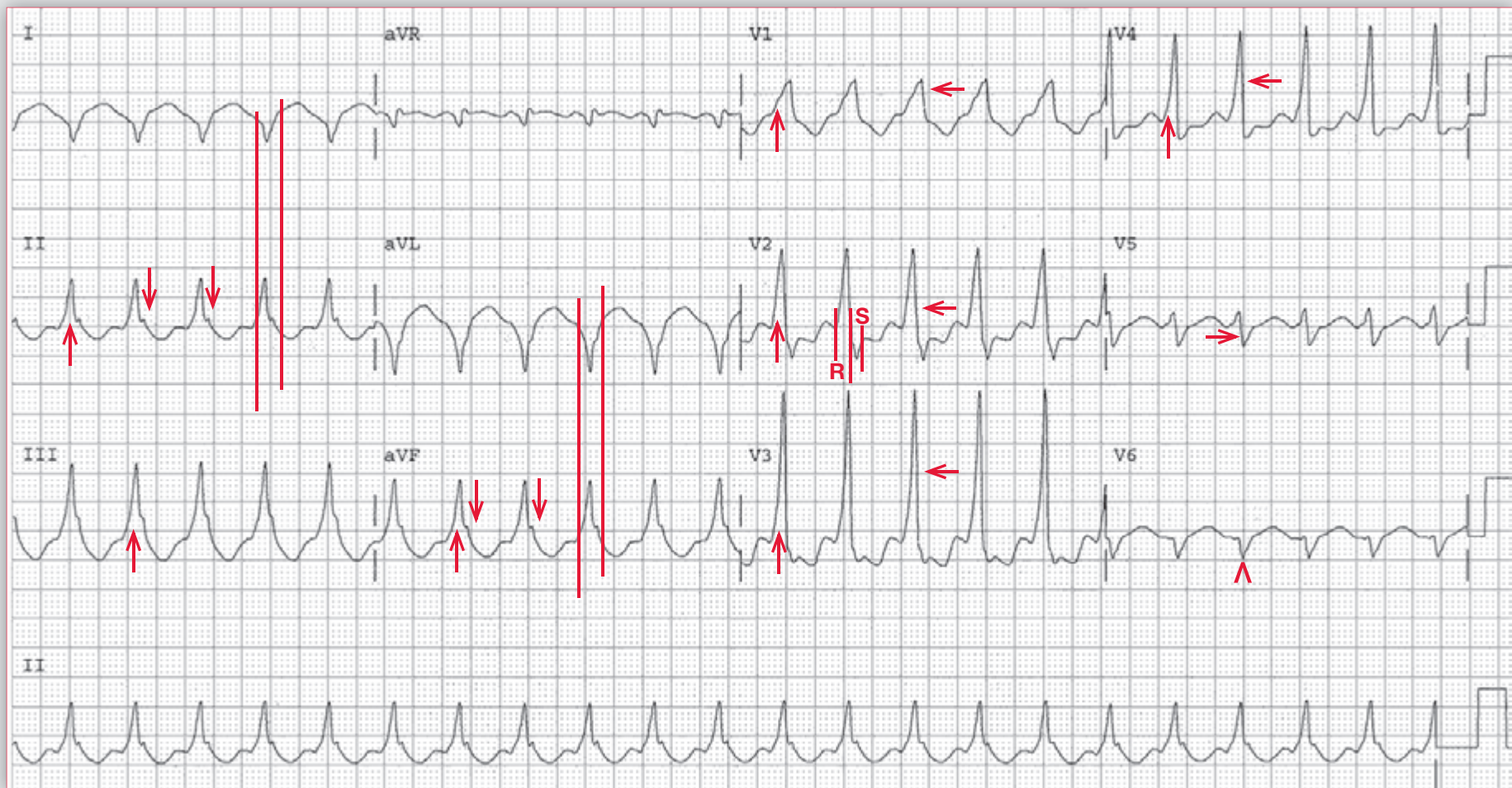
Based on the tracing (ECG 26B), what is the diagnosis and what is the etiology of the arrhythmia?

What therapies can be considered?

ECG 26B



Podrid's Real-World ECGs



ECG 26A Analysis: Wide complex tachycardia, right axis, antidromic atrioventricular reentrant tachycardia

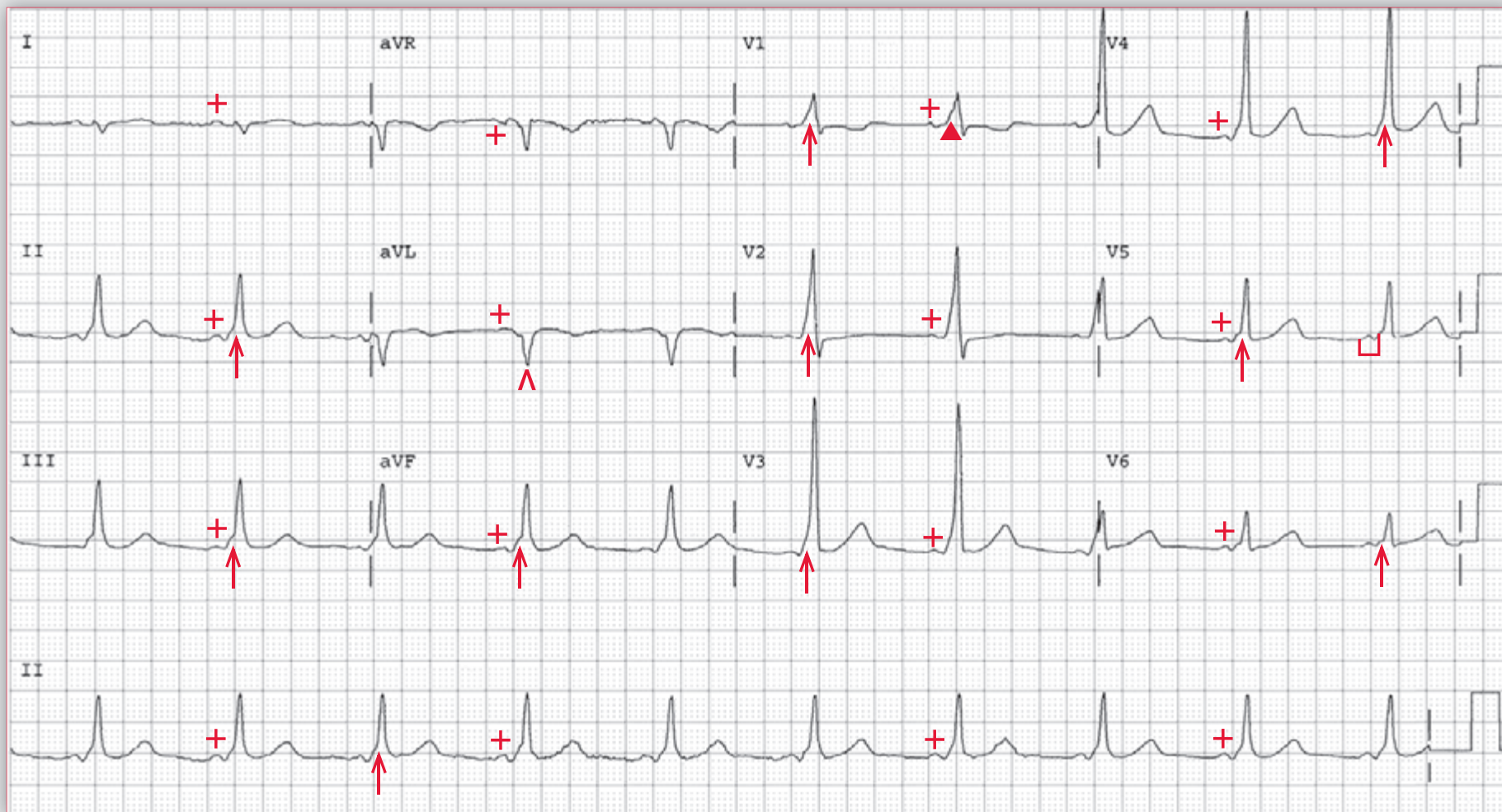
ECG 26A shows regular rhythm at a rate of 136 bpm. The QRS complex duration is increased (0.18 sec). The axis is rightward between $+90^\circ$ and $+180^\circ$ (negative QRS complex in lead I and positive in lead aVF). There are no obvious P waves seen. Importantly the deflection at the end of the QRS complex in leads II and aVF (\downarrow) are not P waves, but are part of the QRS complex. This can be established by determining the maximal QRS duration and comparing it to the QRS complex duration in leads II and aVF (\parallel). The QRS complex has a tall and broad R wave in leads V1–V4 (\leftarrow) while there is a small R wave and deep S wave in lead V5 (\rightarrow) and a QS complex in lead V6 (\wedge). Although resembling a right bundle branch block, the QRS complex morphology is not typical for either a left or right bundle branch block. There are ST-T wave changes (which are consistent) seen in many of the leads.

There are no specific waveforms on the ECG that would be helpful in establishing the etiology of this wide complex tachycardia. The abnormal QRS complex, which has a slurred upstroke (\uparrow), can be seen in any

situation that has direct ventricular myocardial activation and would be consistent with either a ventricular tachycardia or a supraventricular tachyarrhythmia (atrial tachycardia, atrial flutter or antidromic atrioventricular reentrant tachycardia) associated with preexcitation or Wolff-Parkinson-White (WPW). There is a RS complex in lead V2, and although the R wave width is greater than the width of the S wave, which is consistent with a ventricular complex, this may be seen in any situation where there is direct myocardial activation, which includes WPW. The rightward axis is not helpful, nor is the absence of any obvious AV dissociation. The presence of AV dissociation with a wide complex tachycardia establishes the etiology as ventricular tachycardia. AV dissociation cannot be present with a preexcited QRS complex as the accessory pathway begins in the atrium and hence there is always 1:1 atrial to ventricular conduction. However, the absence of obvious AV dissociation is not useful.

continues

Podrid's Real-World ECGs



ECG 26B Analysis: Normal sinus rhythm, Wolff-Parkinson-White pattern, pseudo lateral wall myocardial infarction

ECG 26B is the baseline ECG from the patient in ECG 26A. There is a regular rhythm at a rate of 70 bpm. There is a P wave (+) before each QRS complex, and it has a normal morphology and is positive in leads I, II, aVF, and V5–V6. Hence this is a normal sinus rhythm. The PR interval is short (0.12 sec) (□), and the QRS complex duration is prolonged (0.16 sec) as a result of a slurred upstroke or delta wave (↑). Because of the delta wave, the bottom of the QRS complex is wide, while the top of the QRS complex is narrower. Hence this is a preexcited QRS complex or a Wolff-Parkinson-White pattern. There is a positive delta wave in lead V1 (▲), consistent with a left-sided bypass tract. This was originally called type A (anterior), meaning that the initial impulse was directed anteriorly toward V1 and hence due to a left-sided bypass tract. Type B (back) is associated with a negative delta wave in lead V1, due to the fact that the initial myocardial forces are directed posteriorly, away from V1, as a result of a right-sided bypass tract. In addition, there is a pseudo lateral wall myocardial infarction pattern (Q waves in aVL [^]); this indicates a left lateral bypass tract. The QT/QTc intervals are prolonged (480/520 msec), but are normal when the prolonged QRS complex duration is considered (420/450 msec).

Importantly, the QRS complex in each lead is identical in morphology to the QRS complexes during the tachycardia, establishing the wide complex tachycardia as an antidromic atrioventricular reentrant tachycardia (AVRT). This is the most important way to establish that this wide complex tachycardia is an antidromic AVRT. With an antidromic AVRT, the depolarization or activation of the ventricular myocardium is the result of the impulse conducted antegradely to the ventricle via

the accessory pathway. Retrograde conduction back to the atrium is via the normal His-Purkinje system and AV node. Therefore, the QRS complexes during the tachycardia are wide, resembling the preexcited complexes during sinus rhythm, but they are wider, or maximally preexcited, as all of ventricular myocardial activation is via the accessory pathway. In contrast, during sinus rhythm the preexcited QRS complex is due fusion between the impulse conducted to the ventricles via the accessory pathway and the impulse conducted via the AV node–His-Purkinje system. When ventricular activation or depolarization results from an impulse conducted via the normal AV node–His-Purkinje system and retrograde conduction back to the atrium is via the accessory pathway, the QRS complex is narrow and is supraventricular in morphology. This is termed an orthodromic AVRT. In an AVRT, regardless of whether it is antidromic or orthodromic, the AV node is a part of the circuit and hence any alteration of AV nodal conduction properties may terminate the arrhythmia. Therapies include any vagal maneuver (Valsalva, carotid sinus pressure, coughing), adenosine, a β -blocker, a calcium-channel blocker (verapamil or diltiazem), or digoxin. When there is an orthodromic AVRT and a narrow QRS complex, this is not problematic. However, in the presence of a wide QRS complex, it may be difficult to distinguish between an antidromic AVRT or ventricular tachycardia. If the diagnosis of an antidromic AVRT is definitively established, the treatment is to alter the AV nodal properties, and the same therapies that are used for an orthodromic AVRT are appropriate. The important issue is establishing the diagnosis as it may be challenging to differentiate between an antidromic AVRT and ventricular tachycardia. ■

Core Case 27

A 42-year-old man is brought to the local emergency department after an episode of presyncope. He is currently hemodynamically stable and conscious. He is able to describe the circumstances that brought him to hospital.

He was driving his car when he suddenly felt lightheaded. He was able to apply the vehicle brakes before almost losing consciousness.

His speed was minimal. He denies any pain consistent with blunt force trauma. Airbags did not deploy.

Such symptoms of presyncope have occurred three or four times over the past year, but he has never lost consciousness. His medical history is notable for hypertension, for which he takes a β -blocker.

ECG 27A



During his evaluation, triage vital signs note a tachycardia. You request an ECG (ECG 27A).

Your medical student asks you if a calcium-channel blocker would be appropriate therapy for this patient based on his interpretation of the ECG.

After administering the appropriate therapy, the tachycardia terminates and a repeat ECG is obtained (ECG 27B).

What is your interpretation of tracing (ECG 27A)?

Does it suggest a cause for the patient's clinical presentation?

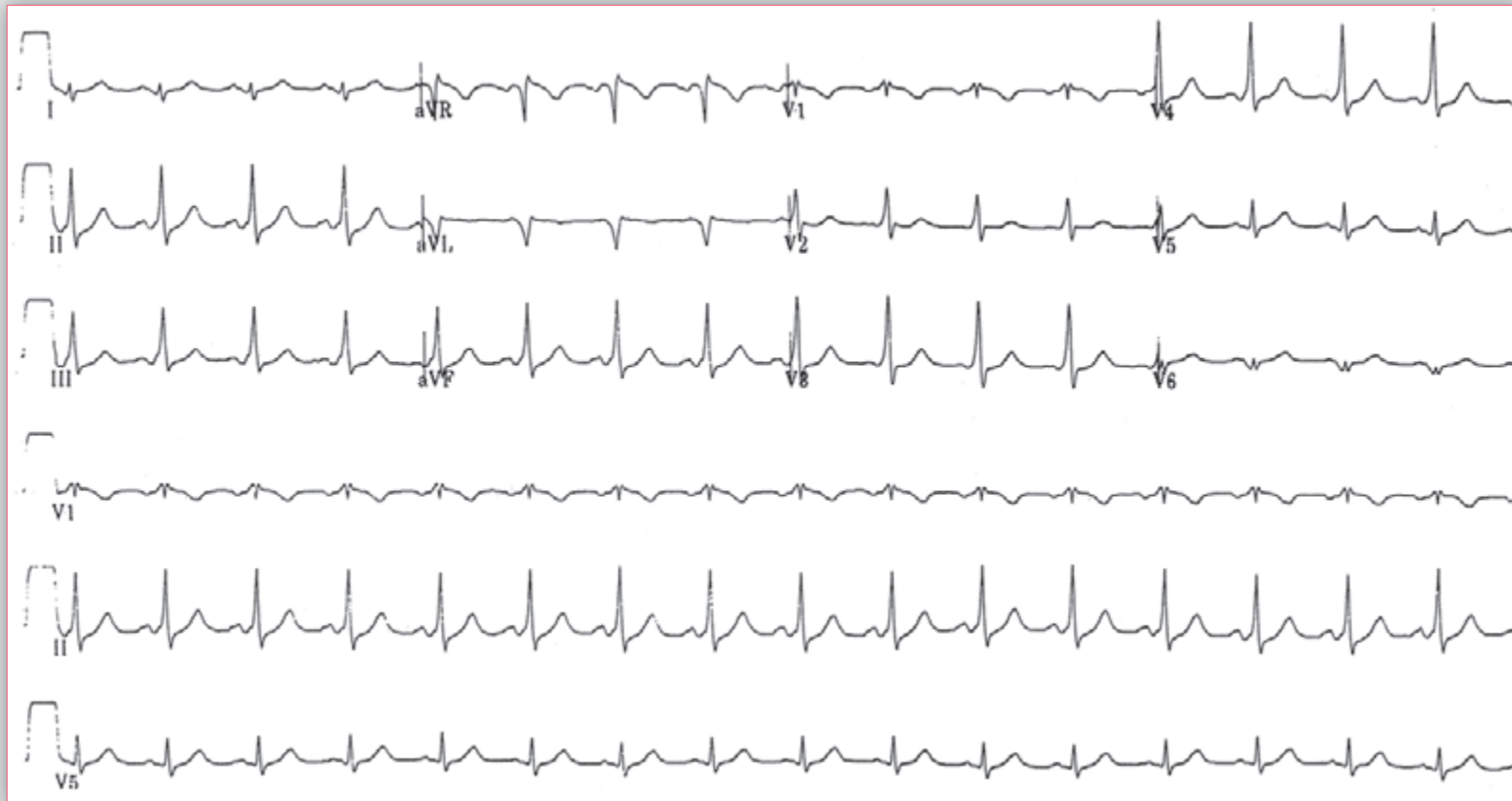
Do you agree with a calcium-channel blocker?

If not, what therapy would you suggest?

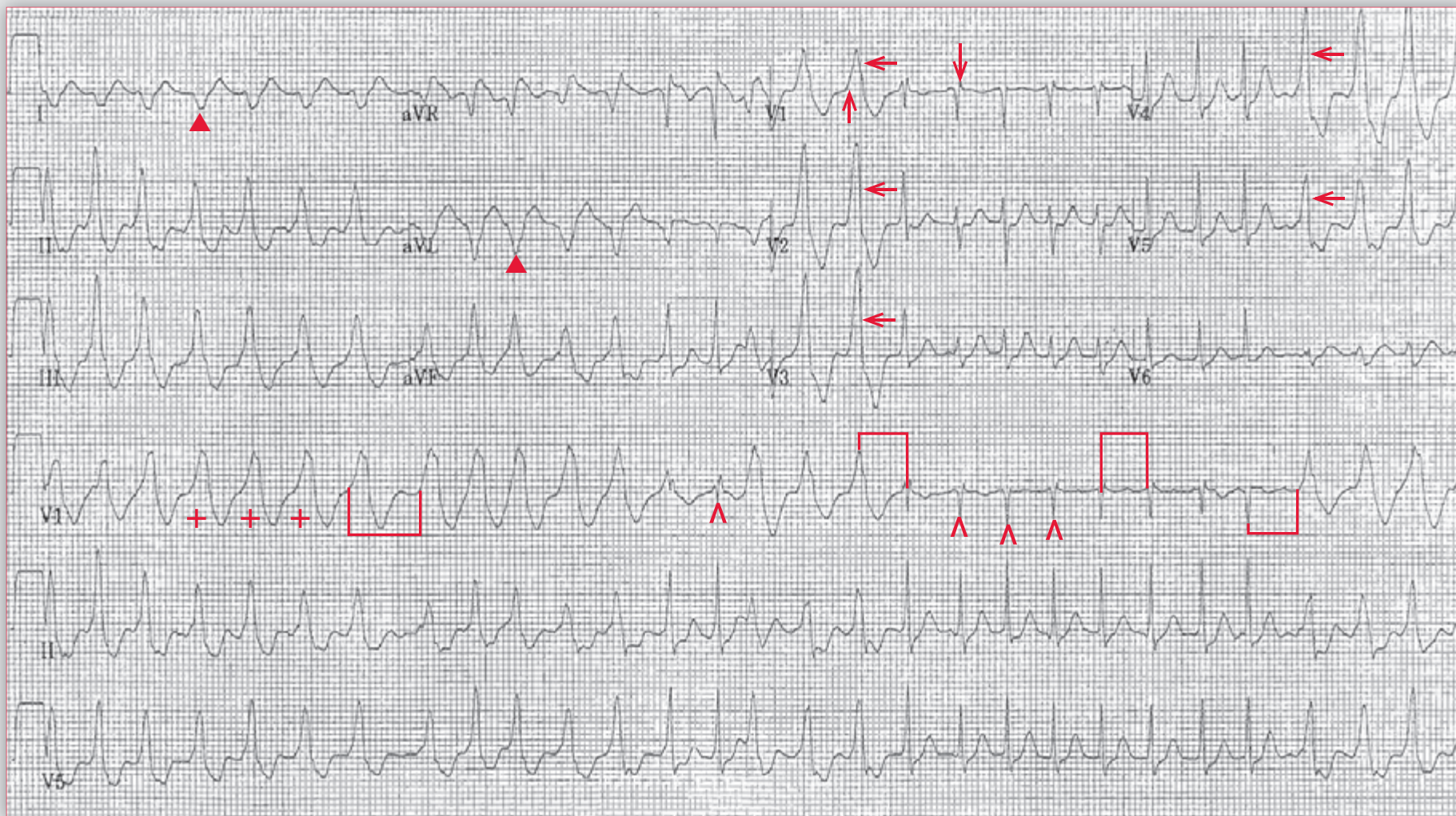
What diagnosis is confirmed in tracing (ECG 27B)?

What was the likely cause of the patient's presyncope?

ECG 27B



Podrid's Real-World ECGs



ECG 27A Analysis: Atrial fibrillation, Wolff-Parkinson-White syndrome

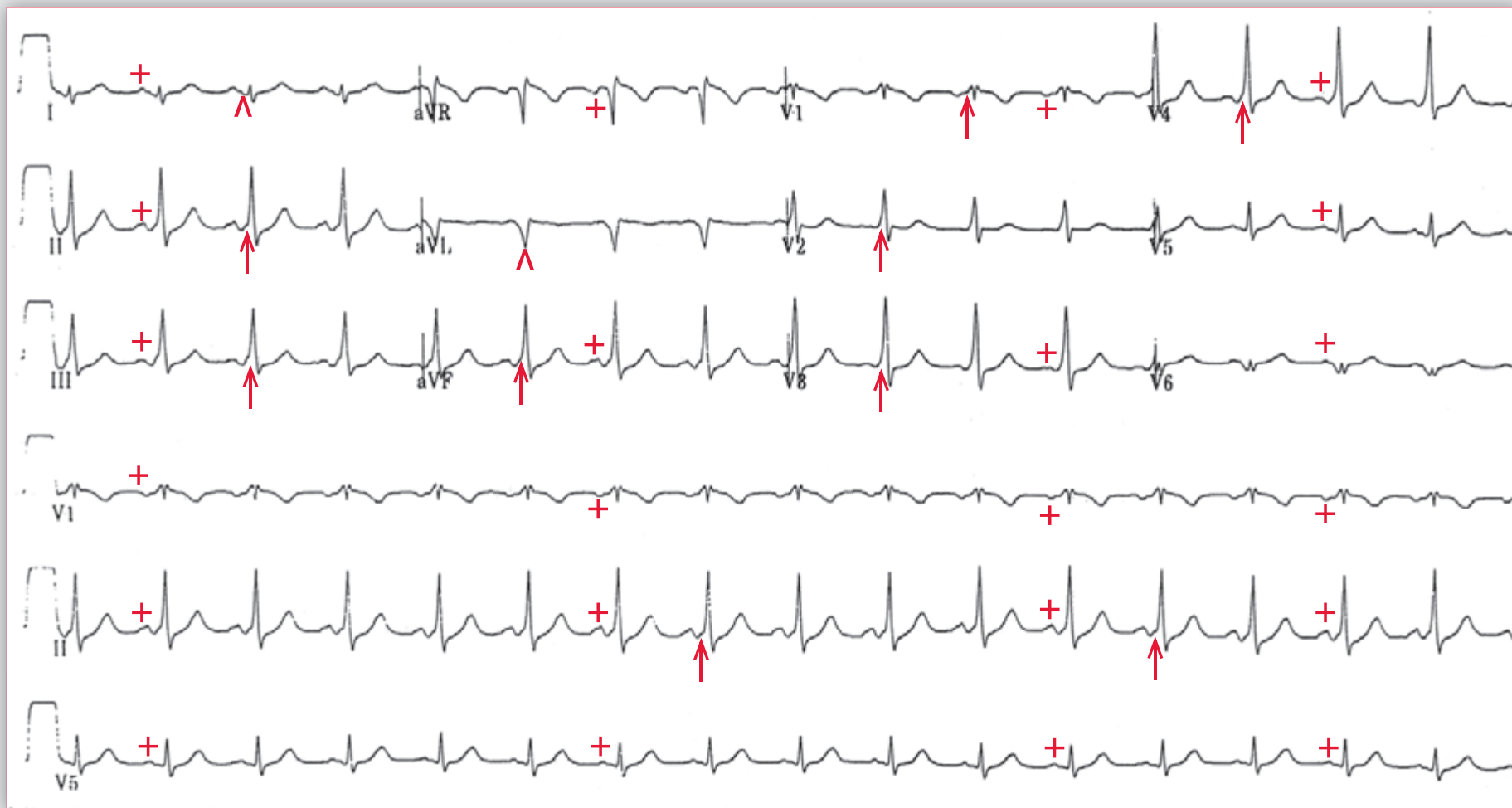
ECG 27A shows the rhythm is irregularly irregular at a rate of 180 bpm. There are only three supraventricular rhythms that are irregularly irregular, including sinus arrhythmia (one P-wave morphology and stable PR interval); multifocal atrial rhythm if the rate is < 100 bpm or multifocal atrial tachycardia if the rate > 100 bpm (> 3 P-wave morphologies with no dominant P-wave morphology and different PR intervals); or atrial fibrillation (no organized P waves). Atrial flutter or atrial tachycardia may be irregular, but there is a pattern to the irregularity, based on the degree of AV nodal blockade or conduction (*ie*, 2:1, 3:1, 4:1 or variable). Therefore, these rhythms may be regularly irregular. There are no organized P waves seen before or after any of the QRS complexes. Hence this is atrial fibrillation. Noted are QRS complexes that are wide (+) and narrow (^). The wide QRS complexes vary in width (0.12 to 0.16 sec) and amplitude. The wide QRS complexes have a right bundle branch block morphology with positive concordance (tall R waves) across the precordium (except for V6) (←). The axis of the wide QRS complexes is rightward between +90° and +180° (negative in lead I and positive in lead aVF).

The narrow QRS complexes have a normal duration (0.08 sec), and the morphology in the precordial leads (where the narrow complexes are seen in each lead) is normal, with a R' in lead V1 (↓), which is due to a minor conduction abnormality to the right ventricle. The most striking finding is a lack of relationship between rate (or RR interval) and QRS duration. Noted is that the RR intervals of the narrow complexes are

shorter (□) than some of the RR intervals where the QRS complexes are wide and aberrated (⊐). Normally, aberration that is rate-related (due to underlying conduction system abnormality) is associated with a QRS duration that is increased when the RR intervals are shorter. The one condition in which there is no relationship between rate (RR interval) and QRS duration is Wolff-Parkinson-White (WPW). With WPW, there is an accessory pathway that serves as a second pathway between atrium and ventricle, in addition to the normal AV node–His-Purkinje pathway. The atrial impulses may conduct only via the AV node–His-Purkinje system and will be narrow in duration; they may conduct only via the accessory pathway and will be very wide as they are maximally preexcited (with the widened QRS complex resulting from a delta wave which is due to direct myocardial activation); or they may conduct via both pathways, resulting in a fusion complex with various degrees of QRS complex widths (or delta wave duration). The fusion complex is due to initial ventricular activation via the accessory pathway as well as activation via the normal AV node–His-Purkinje system. The extent of fusion (resulting in variability in the delta wave and QRS width) is dependent upon the balance of conduction through the two pathways and this is determined by the rate of AV nodal conduction. It is also determined by the location of the atrial impulse origin relative to the two different pathways. The rate of conduction through

continues

Podrid's Real-World ECGs



ECG 27B Analysis: Normal sinus rhythm, Wolff-Parkinson-White pattern

the accessory pathway is constant as this tissue is the same as His-Purkinje tissue. If AV nodal conduction is rapid, only a small amount of myocardium is activated via the accessory pathway and hence the delta wave is narrower. If AV nodal conduction is slower, more of the ventricular activation occurs via the accessory pathway and the delta wave is wider. Atrial fibrillation is associated with variable and irregular conduction through the AV node, based on the rate of AV nodal stimulation as well as the location of the atrial fibrillatory impulse. There will be a variable amount of fusion with variable widths of the QRS complex and delta wave.

The QRS complex in leads I and aVL has a QS morphology (▲) and there is a positive delta wave in lead V1 (↑). Hence this is a left lateral bypass tract. This is termed pseudo lateral wall infarction, as in WPW; abnormalities of the left ventricle cannot be reliably diagnosed because initial ventricular activation is directly through the myocardium as a result of early activation via the accessory pathway.

ECG 27B is from the same patient as ECG 27A. There is a regular rhythm at a rate of 96 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.12 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration is prolonged (0.14 sec) as a result of a broad and slurred upstroke (↑), which is the delta wave. As a result of the delta wave the bottom of the QRS complex is wide, while the top of the QRS complex is narrow. Hence the QRS complexes have a Wolff-Parkinson-White pattern. There is a Q wave in leads I and aVL (^) and positive delta wave in lead V1 (↑), consistent with a left lateral accessory pathway. The QRS complex morphology is very similar to what is seen in ECG 27A, although most of the QRS complexes in ECG 27A are even wider as they are more preexcited, indicating that most of the QRS complexes during atrial fibrillation are being conducted to the ventricle via the accessory pathway. The QT/QTc intervals are long (400/510 msec) but are normal when the widened QRS complex is considered (360/455 msec).

continues

It is important to recognize WPW in patients with atrial fibrillation with a rapid ventricular response rate, as AV nodal blocking drugs, which are routinely given to slow the ventricular response rate in atrial fibrillation, should not be used with WPW because of the potential to accelerate the ventricular rate to greater than 350 to 450 bpm (which is atrial fibrillatory rate), which increases the potential to provoke ventricular fibrillation. As WPW represents fusion between conduction via two separate pathways, if the AV node is blocked, then all of ventricular activation is via the accessory pathway. If this pathway has a short refractory period, it is capable of very rapid stimulation and hence will conduct to the ventricle at very rapid rates (as are seen with atrial fibrillation) that can result in the precipitation of ventricular fibrillation.

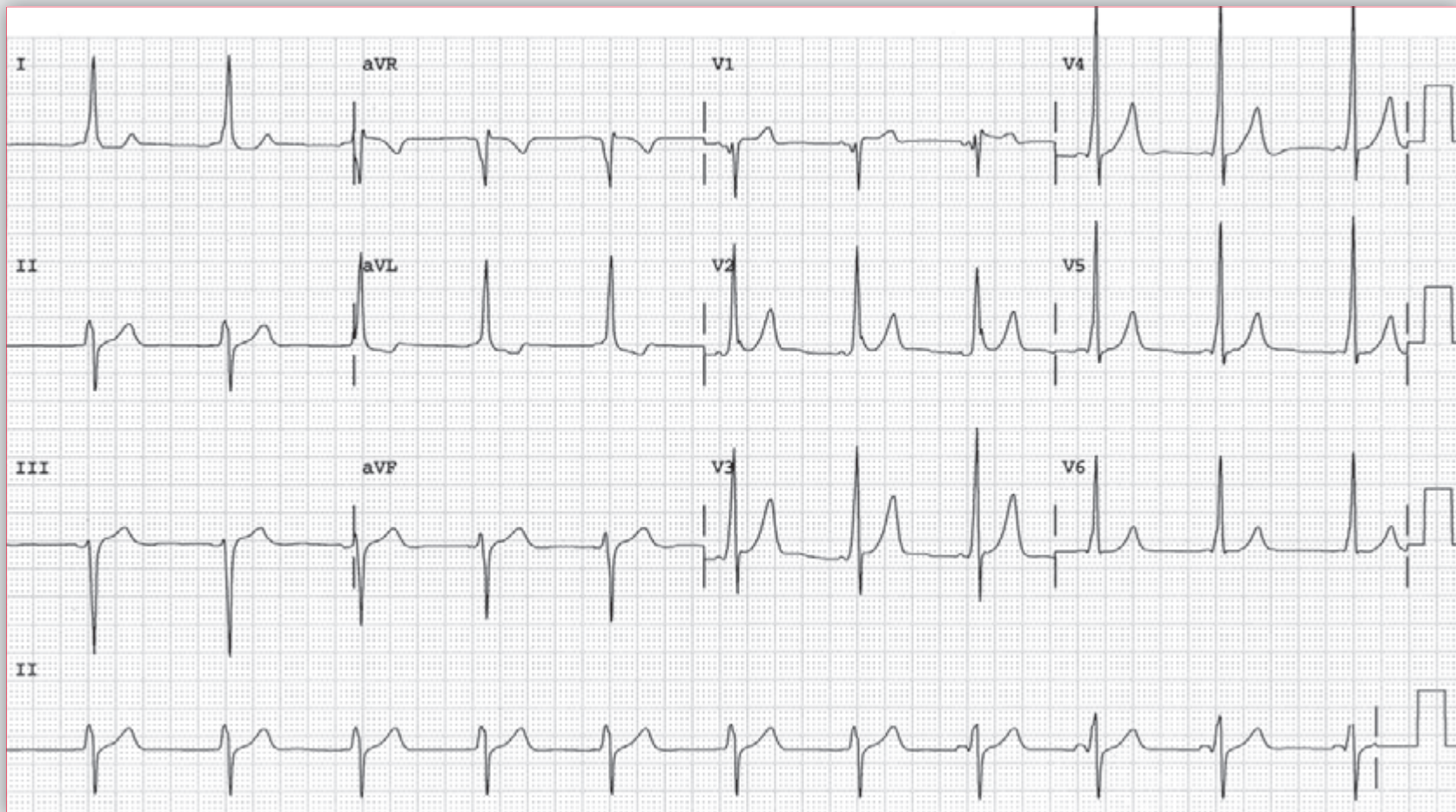
In addition, there is the loss of the modulating effect of impulse conduction via the normal pathway. Impulses coming through the normal His-Purkinje system can intermittently enter the accessory pathway retrogradely. This retrograde conduction can prolong the refractoriness of the accessory pathway (retrograde concealed conduction), which will reduce the ability of this pathway to conduct at rapid rates.

Acutely, appropriate therapy consists of intravenous antiarrhythmic drugs that slow conduction of impulses via the bypass tract. These agents include IV procainamide or IV ibutilide. These agents also have the potential to revert atrial fibrillation to sinus rhythm. ■

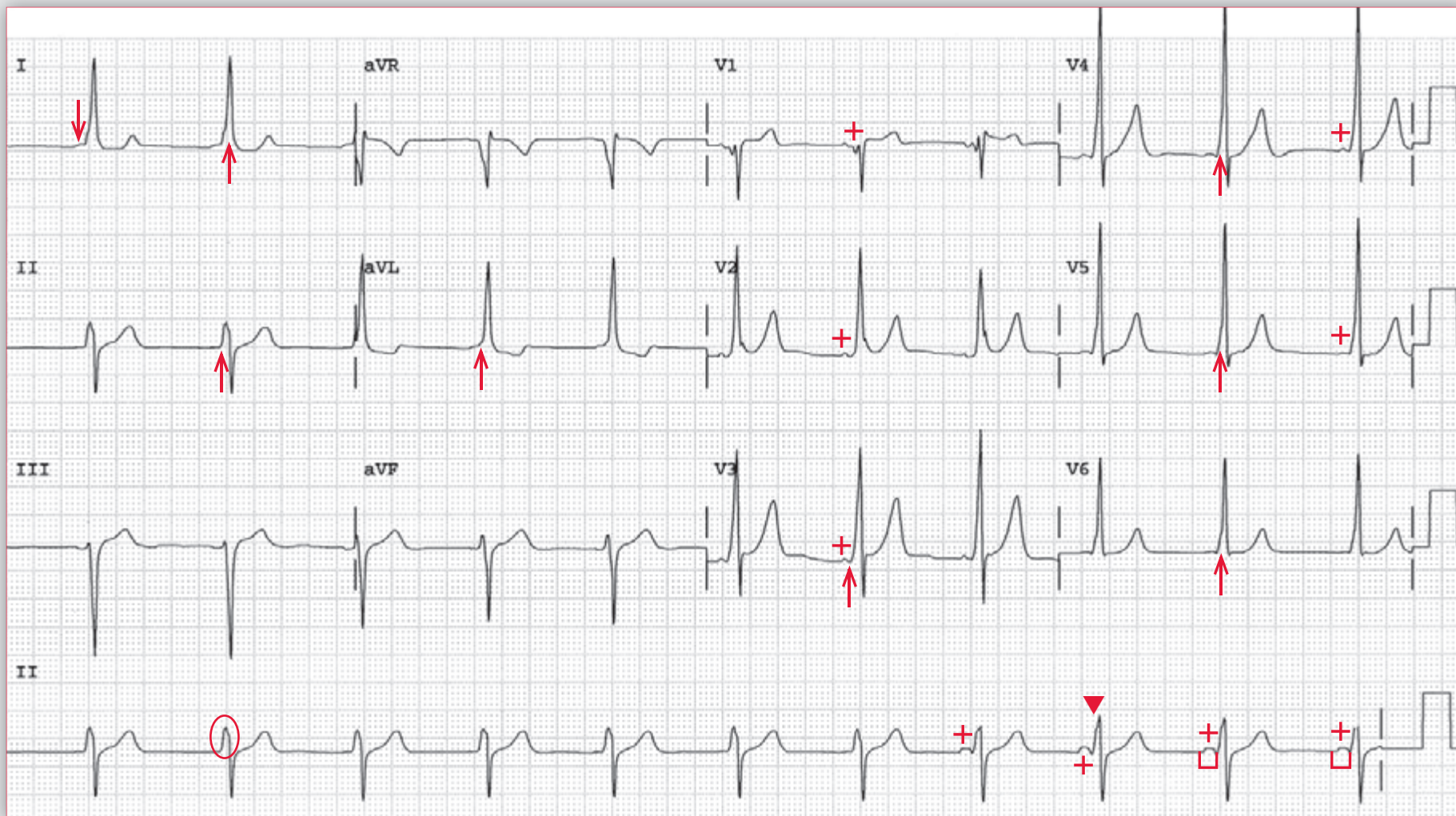
A 25-year-old man is seen by his physician for a routine physical examination. He denies any medical history and his only complaint is occasional palpitations. He is fully active. As part of his evaluation, an ECG is obtained. The ECG causes concern, and as a result the patient is referred urgently to a cardiologist.

What does the ECG show and what is the reason for concern?

Is any additional therapy needed?



Podrid's Real-World ECGs



ECG 28 Analysis: Normal sinus rhythm, Wolff-Parkinson-White pattern, concertina effect

There is a regular rhythm at a rate of 72 bpm. The QRS complex duration is increased (0.14 sec). This is due to the slurred and prolonged upstroke of the QRS complex (↑). This is a delta wave. There are no clear P waves seen in the initial part of the ECG (as noted in the lead II rhythm strip), although there are P waves that can be seen in lead I which are merged with the QRS complex (↓). P waves (+) can be seen before QRS complexes 8–11. The PR interval in these complexes is stable, but very short (0.11 sec) (⊐). The P waves are positive in leads II and V4–V6. Hence this is probably a normal sinus rhythm. The short PR interval and delta wave is diagnostic of a Wolff-Parkinson-White (WPW) pattern. The QT/QTc intervals are prolonged (440/480 msec) but are normal when the prolonged QRS complex duration is considered (400/440 msec).

It can be seen that in the first part of the lead II rhythm strip, where P waves are absent, the delta wave is slightly wider (○), while in the latter part of the lead II rhythm strip, where P waves are seen, the delta wave is narrower (▼). This change in the PR interval and delta wave width can occasionally be seen in WPW and is referred to as a concertina effect. This effect occurs as a result of changes in AV nodal conduction relative to conduction via the accessory pathway. The WPW pattern represents fusion between initial or early ventricular myocardial activation (preexcitation) resulting from impulse conduction via the accessory pathway (which has the same characteristics as

the His-Purkinje system and has a conduction velocity that is fixed regardless of the heart rate, *ie*, conduction is all or none) followed by ventricular myocardial activation resulting from impulse conduction through the normal AV nodal–His-Purkinje pathway. Conduction velocity through the AV node is variable, depending upon local factors as well as autonomic tone. As the QRS complex represents a balance of conduction between these two pathways, any change in conduction through the AV node will result in changes in the amount of preexcitation or myocardial activation via the accessory pathway. If AV nodal conduction velocity is increased, less myocardial activation results from accessory pathway activation. The PR interval is longer and the delta wave less wide. A decrease in AV nodal conduction will result in more myocardial activation via the accessory pathway and hence a shorter PR interval and wider delta wave. The change in AV nodal conduction may occur as a result of local effects on the AV node or changing autonomic (parasympathetic and sympathetic) tone, which is independent of autonomic effects on the sinus node; hence there may not be any change in sinus rate.

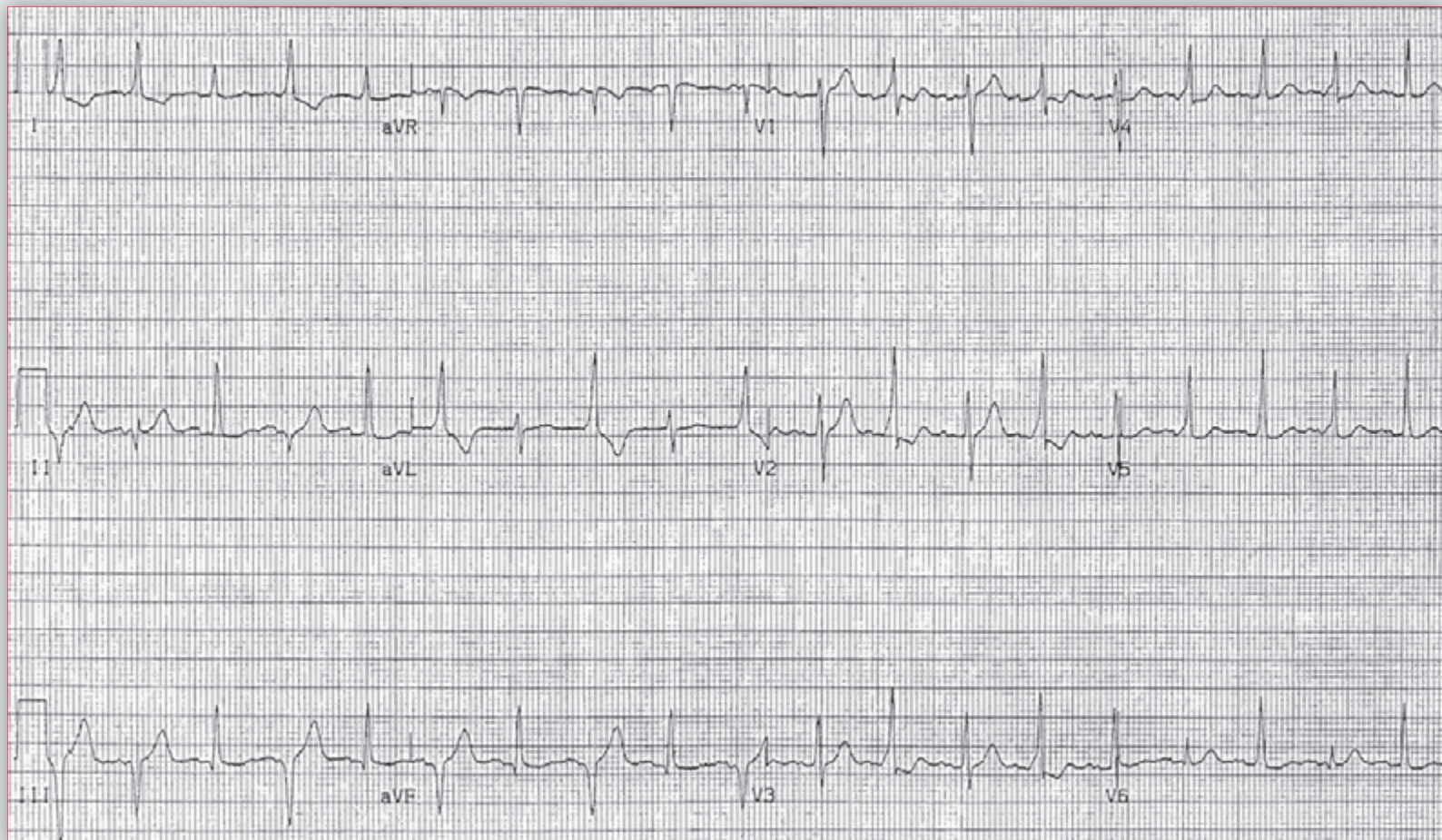
The physician's concern based on the ECG is likely related to the initial absence of P waves and the potential that this is a ventricular rhythm, based on the wide and abnormal QRS complexes. Thereafter the short PR interval with the wide QRS complex might be considered to be a ventricular rhythm with AV dissociation. ■

Core Case 29

A 17-year-old male is admitted to hospital with a traumatic ACL tear. After surgical repair of his ligament, he is being monitored on the orthopedic ward. He suddenly complains of palpitations. His heart rate is noted to be elevated. An ECG is obtained (ECG 29A).

Shortly thereafter, he suddenly complains of lightheadedness and appears in distress. His heart rate is markedly elevated, and his blood pressure falls. A repeat ECG is obtained from his telemetry monitors (ECG 29B).

ECG 29A



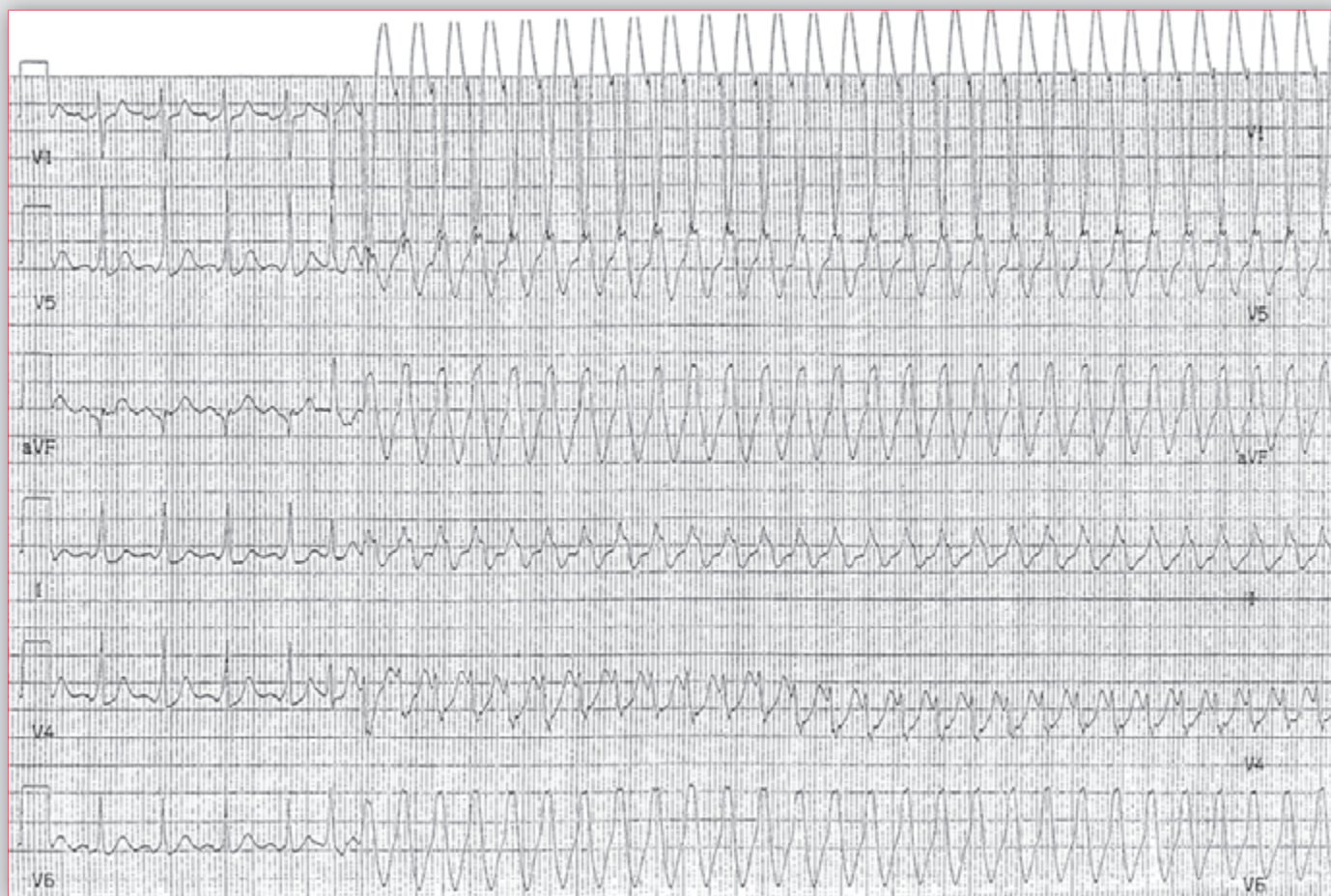
What is your interpretation of the tracing (ECG 29A)?

What arrhythmic pattern does this represent?

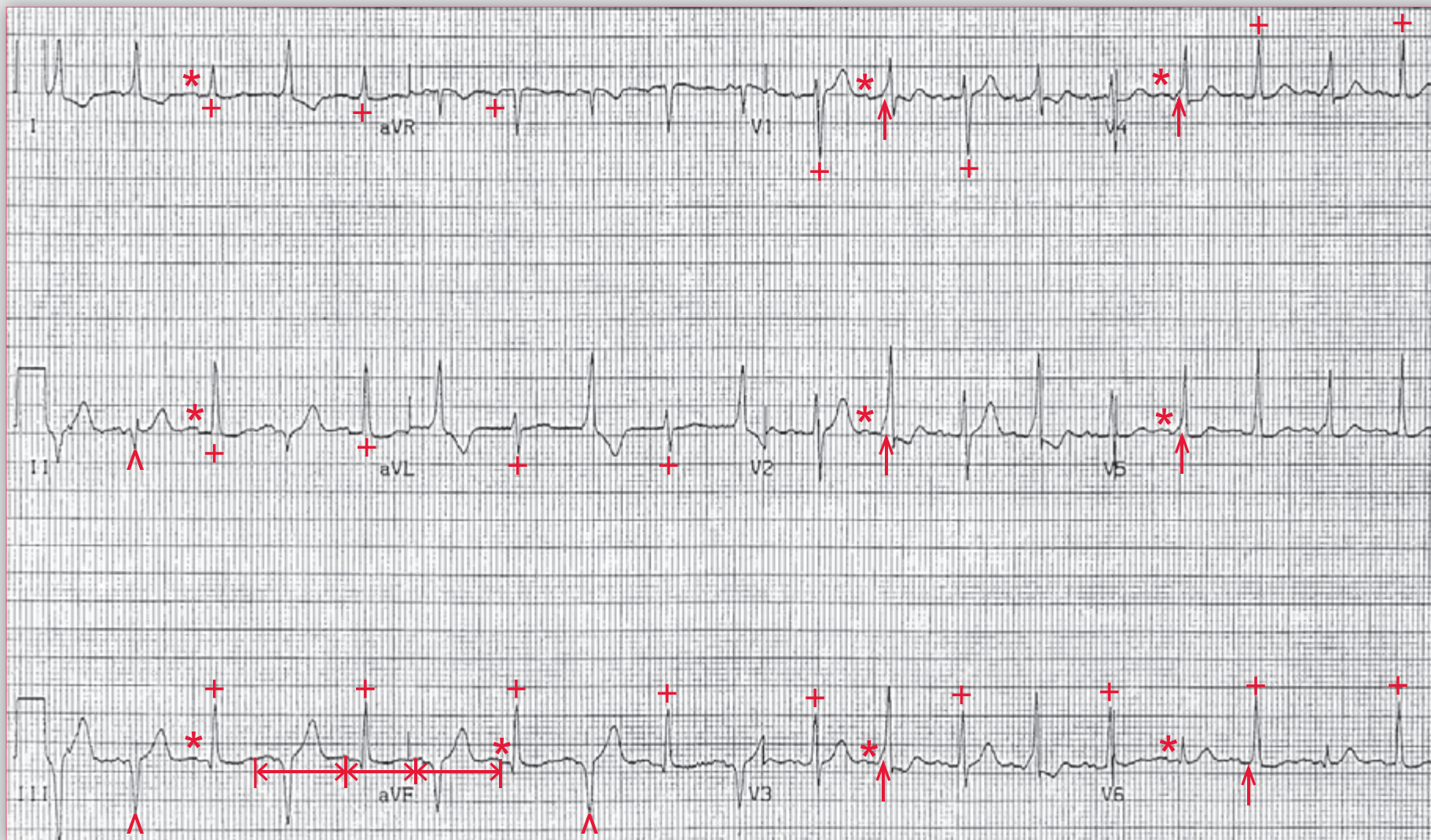
What diagnosis can be made based on the second tracing (ECG 29B)?

What therapy is appropriate?

ECG 29B



Podrid's Real-World ECGs



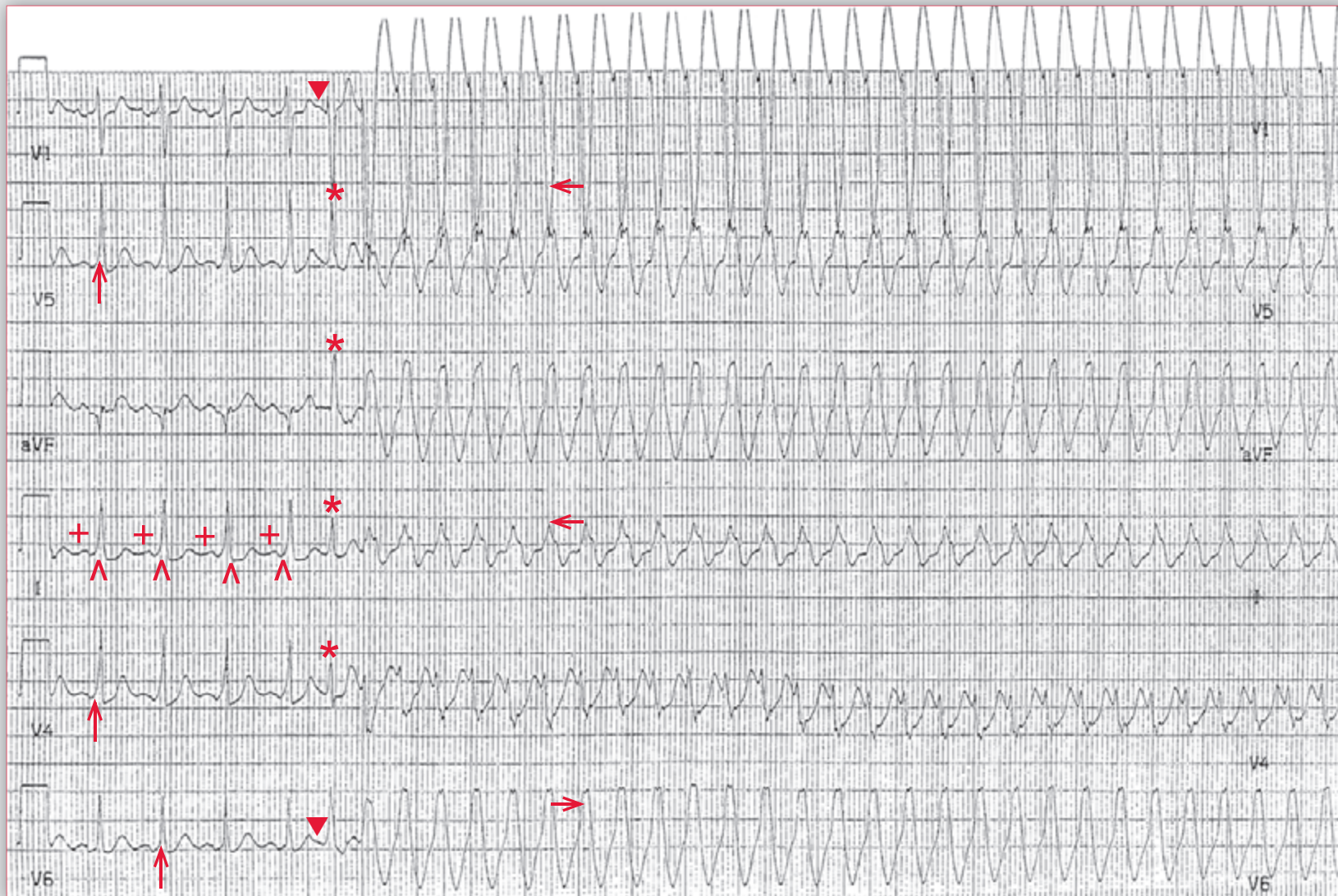
ECG 29A Analysis: Sinus tachycardia, left atrial hypertrophy (abnormality), intermittent Wolff-Parkinson-White pattern

ECG 29A shows a regular rhythm at a rate of 120 bpm. There are two different QRS complex morphologies and durations that occur in a repeating fashion. Every other QRS complex is narrow (+) with a normal duration (0.08 sec) and axis between 0° and $+90^{\circ}$ (positive QRS complex in leads I and aVF). The alternating QRS complex is wider (duration 0.14 sec) and has an extreme leftward axis between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF). The negative QRS complexes in leads II and aVF (as well as lead III) is due to deep Q waves (^), consistent with an inferior wall myocardial infarction. These QRS complexes are wide (primarily at the base of the complex) as a result of a slow and slurred upstroke, best seen in leads V1–V6 (↑). This is a delta wave and it is consistent with a Wolff-Parkinson-White (WPW) pattern. Thus, these complexes are preexcited. The QT/QTc intervals (of the narrow complexes) are normal (300/420 msec).

There are P waves seen before each QRS complex (*) and the PP interval is constant (↔). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. However, there are two different PR intervals. The PR interval associated with each of the narrow QRS complexes is 0.22 second, representing a first-degree AV block

(prolonged AV conduction). The PR interval associated with the pre-excited QRS complex is shorter, but still normal (0.14 sec). The PR interval with the preexcited complex should be short. In this case, the PR interval is normal because of a widened and abnormal P wave, which has prominent notching (P-mitrale), seen best in leads V3–V6. The PR interval represents conduction through the atria as well as the AV node–His-Purkinje system. The PR segment is actually a measure of AV nodal–His-Purkinje system conduction. Hence in WPW there is a bypass of the AV node–His-Purkinje system, and the shortened PR interval is actually due to a short PR segment. In this case, there is indeed no PR segment. This patient has an intermittent WPW pattern (beat-to-beat). The inferior Q waves are actually a pseudo inferior wall infarction pattern and the narrow QRS complexes, which are the result of conduction through the normal AV node–His-Purkinje system, do not have Q waves. In WPW, initial ventricular activation is direct via the accessory pathway. Therefore, abnormalities of the ventricular myocardium cannot be reliably diagnosed. A pseudo inferior wall myocardial infarction pattern indicates that the accessory pathway is posteroseptal in location. The delta wave is positive in lead V1, indicating that the initial activation is directed towards lead V1 (originally termed WPW type A) and is, therefore, a left ventricular pathway.

continues



ECG 29B Analysis: Sinus tachycardia, Wolff-Parkinson-White pattern, premature atrial complex, orthodromic atrioventricular reentrant tachycardia (AVRT) with rate-related left bundle branch block

ECG 29B is from the same patient as ECG 29A. The first four QRS complexes (^) are regular at a rate of 130 bpm. The QRS complex duration is increased (0.14 sec) as a result of a delta wave (†). The morphology of these complexes is the same as the preexcited complexes seen in ECG 29A. There is a P wave (+) before each of these QRS complexes and the PR is 0.14 second, similar to the PR interval associated with the preexcited complexes seen in ECG 29A. Hence these complexes have a WPW pattern. The fifth QRS complex is premature (*) and narrow, with a morphology similar to the narrow QRS complexes seen in ECG 29A. Although there is no obvious P wave seen before this premature complex, it can be seen that there is a notch on the downstroke of the preceding T wave, most evident in leads V1 and V6 (▼). This is a P wave, and hence this is a premature atrial complex. As it is narrow and not preexcited, it is conducted to the ventricle via the normal AV node–His–Purkinje system. Immediately following this premature complex is a wide complex tachycardia at a rate of 220 bpm. The QRS complex duration is 0.16 second and the morphology is a typical left bundle branch block with a QS complex in lead V1 (←) and a broad R wave in leads I and V6 (→). There are no obvious P waves seen. However, as the arrhythmia is initiated with a premature atrial complex and since there is a typical left bundle branch block pattern seen, this is most likely a supraventricular tachycardia. As there is a WPW pattern present, the most likely rhythm is an atrioventricular reentrant tachycardia (AVRT). The QRS complex is widened, and it might be assumed that this is an antidromic AVRT (*ie*, activation of the ventricles via the accessory pathway accounting for the widened QRS complex with retrograde activation of the atria via the accessory pathway). With an antidromic AVRT, the QRS complexes resulting from activation via the accessory pathway are preexcited and have a

morphology that is identical to the preexcited QRS morphology seen during sinus rhythm, although wider, as they will be maximally preexcited (as all of ventricular activation is via the accessory pathway). In this case, the QRS morphology during the AVRT is very different from the preexcited QRS complexes, *ie*, there is no delta wave and there is a typical left bundle branch block morphology. Hence this is an orthodromic AVRT with a rate-related left bundle branch block, as in ECG 29A there are narrow sinus QRS complexes that are not aberrated. In an orthodromic AVRT, the antegrade conduction to the ventricles is via the normal AV node–His–Purkinje system, while the retrograde atrial activation is through the accessory pathway. Further supporting that this is an orthodromic AVRT is the fact that the arrhythmia was initiated by a premature atrial complex that was narrow and, therefore, conducted to the ventricles via the normal AV node–His–Purkinje system. Thus, there had to be retrograde atrial conduction via the accessory pathway in order to complete the circuit and precipitate the reentrant arrhythmia.

The acute treatment for terminating an AVRT (antidromic or orthodromic) is a vagal maneuver (Valsalva, carotid sinus pressure, coughing) or an AV nodal blocking agent that will slow or block conduction through the AV node, which is a necessary part of the reentrant circuit and is indeed the weakest link. This includes adenosine, calcium-channel blocker (verapamil or diltiazem), a β -blocker, or digoxin. However, an antidromic AVRT may be difficult to diagnose as it can resemble a ventricular tachycardia. Before treating this arrhythmia with an AV nodal blocking agent, the etiology of the arrhythmia should be definitively established. If the arrhythmia is definitely an antidromic AVRT, an AV nodal blocking agent can be administered. ■

Core Case 30

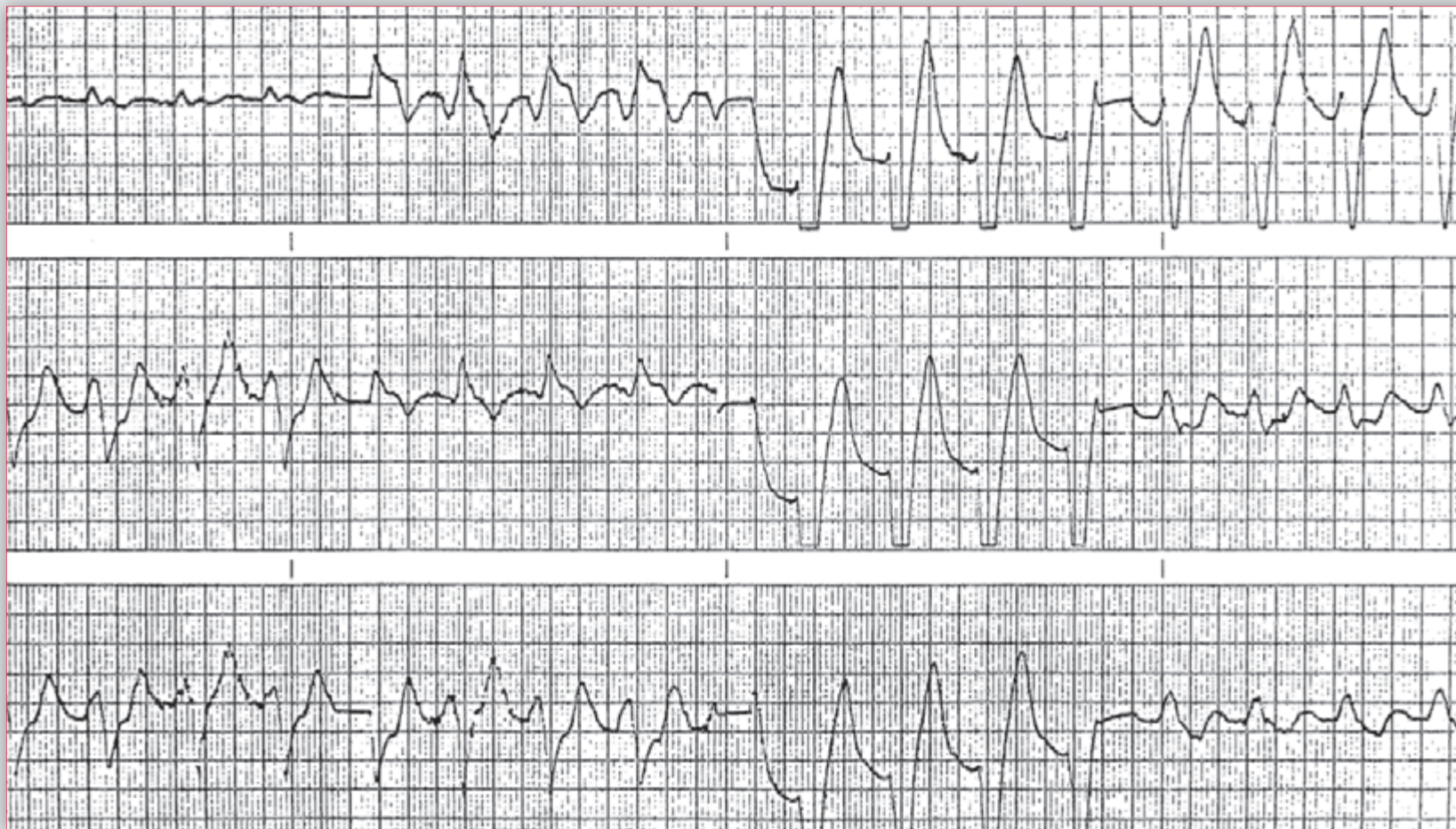
The patient has a history of symptomatic nonsustained ventricular tachycardia being treated with propafenone. During therapy, the baseline ECG is obtained (ECG 30A). The patient

ECG 30A



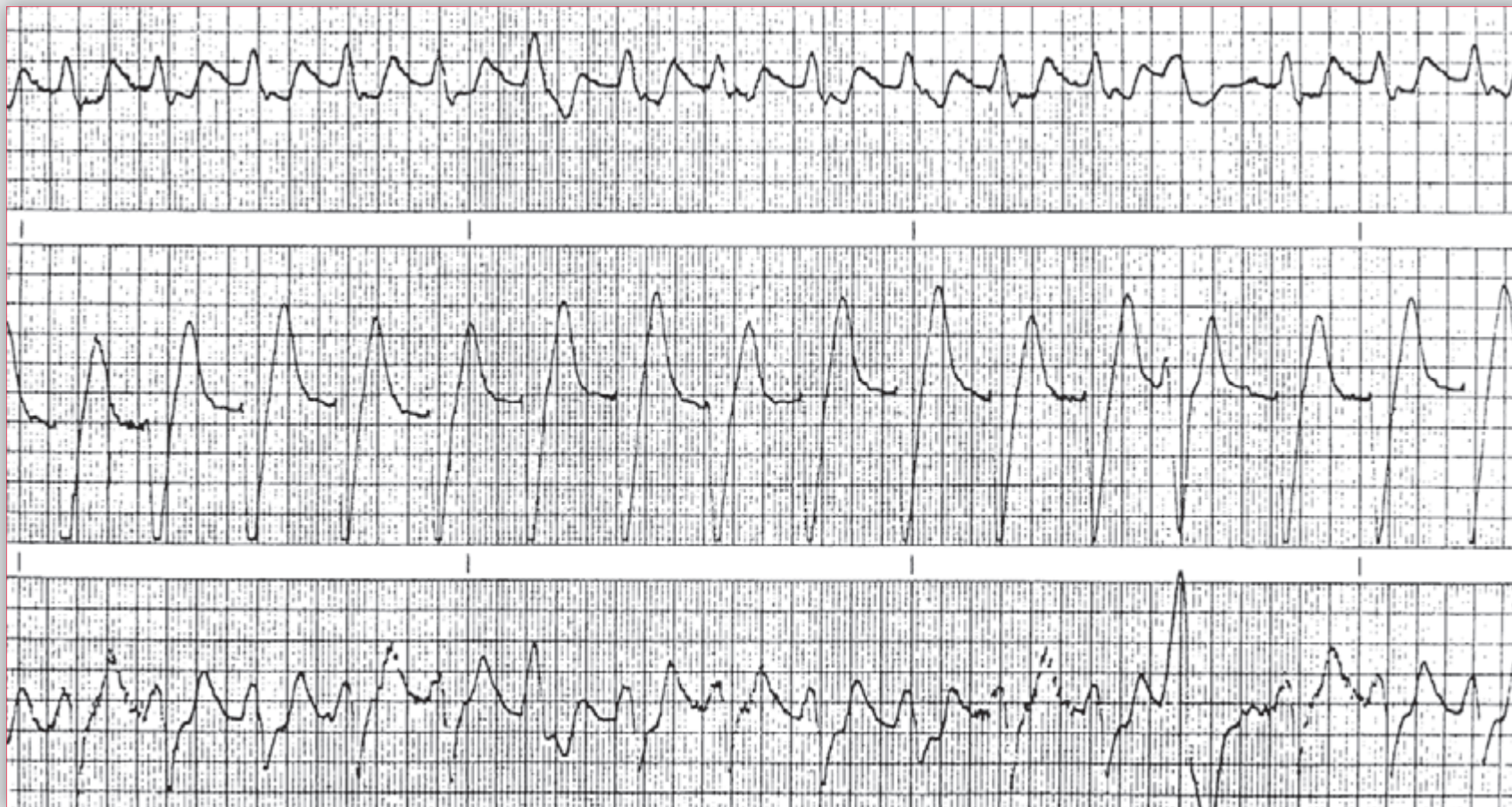
underwent an exercise test. At 3 minutes, ECG 30B was obtained, and the exercise test was abruptly terminated. While being monitored after the exercise, another ECG was recorded (ECG 30C).

ECG 30B



Core Case 30

ECG 30C

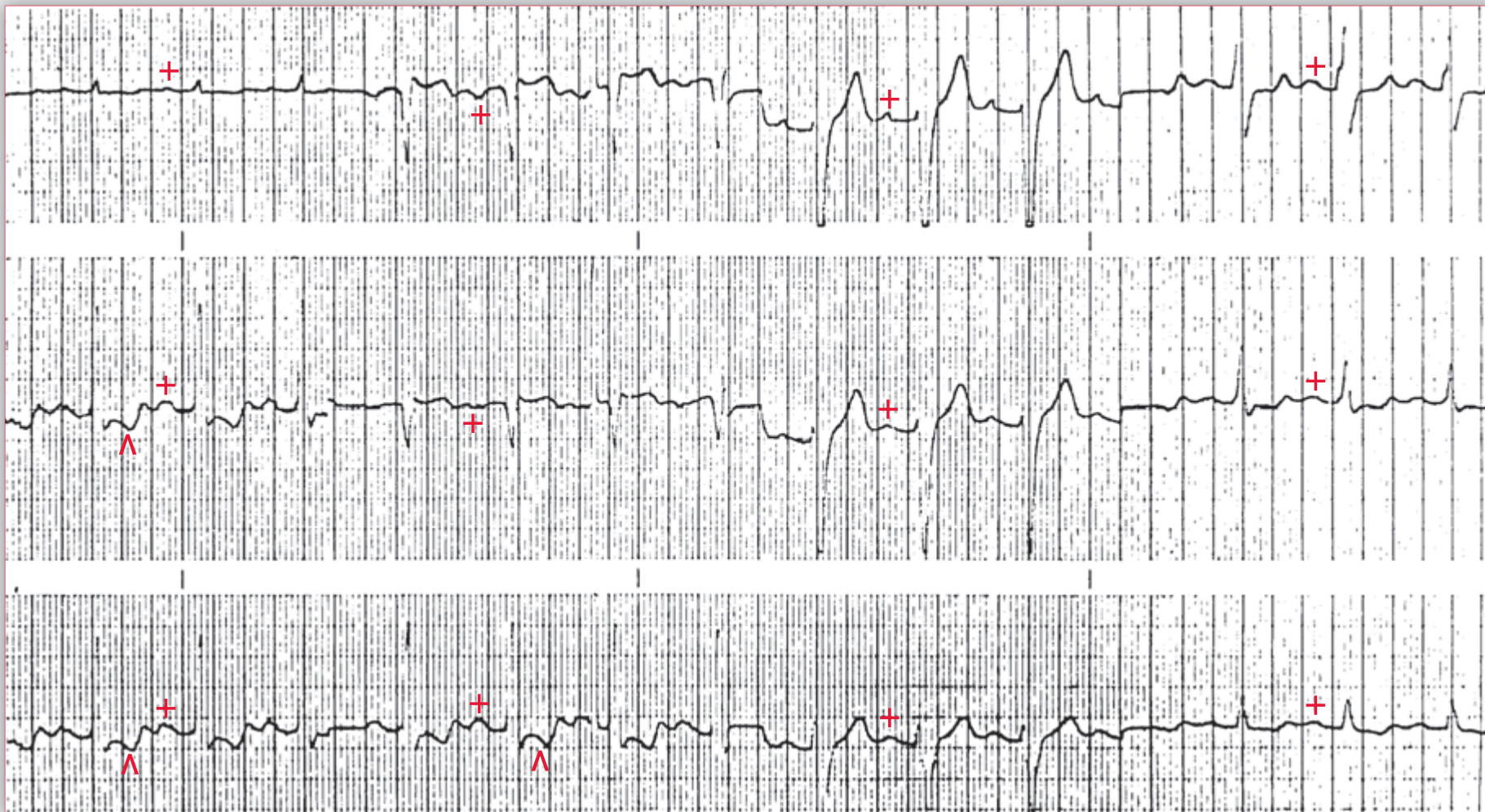


What does ECG 30A show?

What is the rhythm in ECG 30B that resulted in the termination of exercise?

What is the cause for the abnormality seen?

Podrid's Real-World ECGs



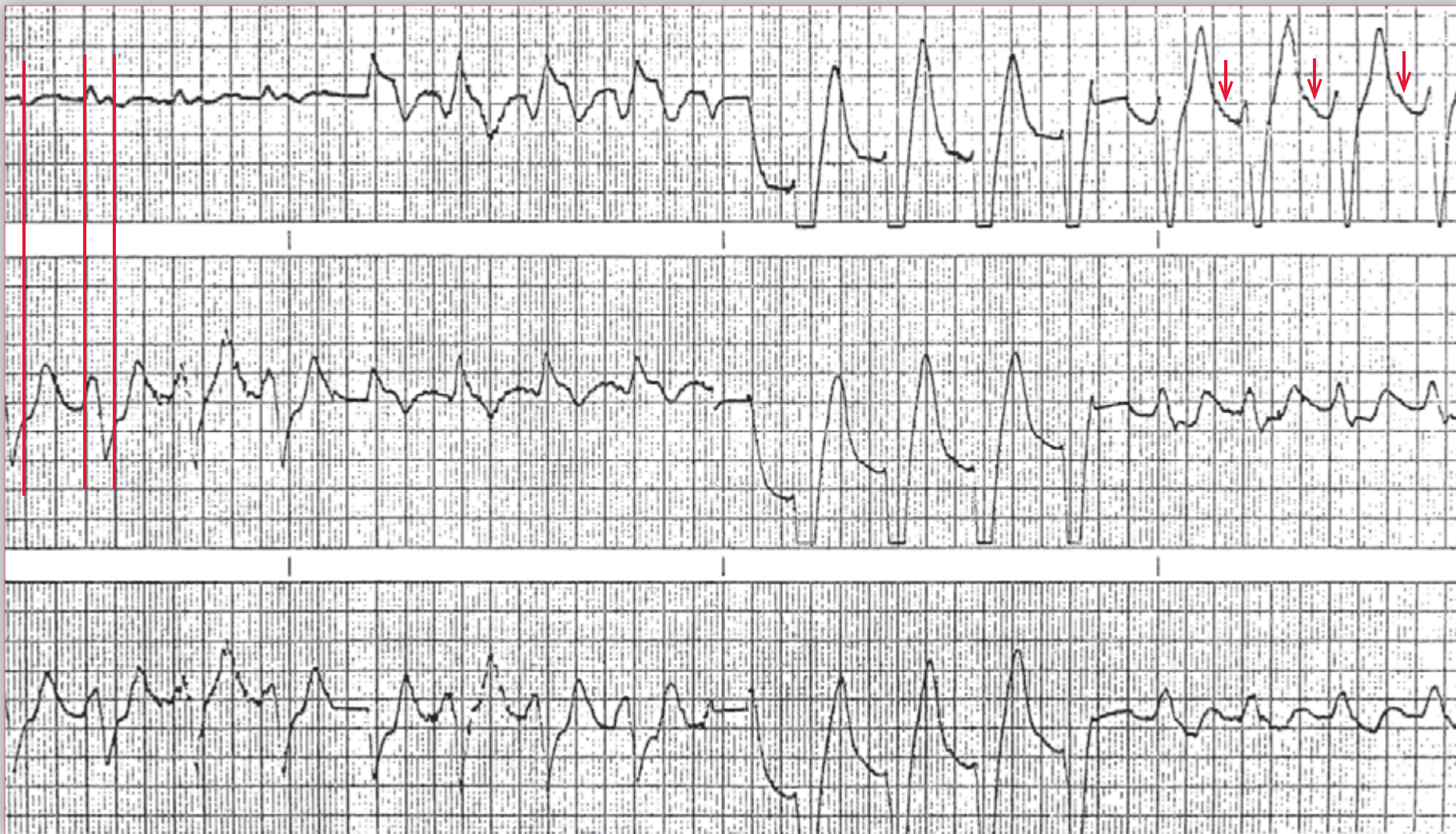
ECG 30A Analysis: Normal sinus rhythm, first-degree AV block (prolonged AV conduction), intraventricular conduction delay, nonspecific ST-T wave abnormalities

ECG 30A shows a regular rhythm at a rate of 90 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.26 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm with a first-degree AV block (prolonged AV conduction). The QRS complex duration is prolonged (0.12 sec) and there is neither a right nor left bundle branch block pattern. Hence this is

an intraventricular conduction delay. The axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). There are also nonspecific ST-T wave abnormalities seen (^), especially in leads II, III, and aVF. The QT/QTc intervals are prolonged (400/490 msec) but are only slightly prolonged when the prolonged QRS complex duration is considered (380/465).

continues

Podrid's Real-World ECGs



ECG 30B Analysis: Sinus tachycardia, first-degree AV block (prolonged AV conduction), intraventricular conduction delay (use-dependent effect of antiarrhythmic drug), nonspecific ST-T wave abnormalities

The patient underwent an exercise test on a treadmill. After 3 minutes, ECG 30B was obtained. There is a regular rhythm at a rate of 100 bpm. The QRS complex duration is wide (0.20 sec) and there are no obvious P waves seen. Although there is a waveform in lead I that looks like a P wave, measurement of the QRS complex duration in another lead (such as lead II) (||) confirms that this waveform is actually part of the QRS complex. There are subtle notches of the downstroke of the T waves (↓), best seen in lead V4, which suggests that there is perhaps a P wave. If so, the PR interval is 0.26 second, which is identical to the PR interval in ECG 30B. The axis is now extremely leftward between -30° and -90° (positive QRS complex in lead I and negative in leads II and aVF). The QT/QTc intervals are long (400/515 msec) but are normal when corrected for the prolonged QRS duration (300/390 msec).

The sudden widening of the QRS complex duration with what was felt to be absent P waves was concerning for sustained ventricular tachycardia, which prompted the discontinuation of the test. The QRS complex has the same morphology as was seen during the baseline ECG 30A, although it is diffusely wider. In addition, with close inspection it can be seen that there are probably P waves present. As a result of the increased heart rate and the baseline first-degree AV block, the P waves merge with the T wave and are not obvious. However, they are seen as a notching of the downslope of the T wave. Importantly, T waves should be smooth in upstroke and downstroke and hence any notching, bump, or irregularity suggests a superimposed P wave. The sudden widening of the QRS complex is a result of the use-dependency of antiarrhythmic drugs, and this is seen most commonly with the class IC antiarrhythmic drugs.

The major effect of the class I antiarrhythmic agents is to interfere with the rapid influx of sodium during phase 0 of the action potential. The rate of sodium influx determines the upstroke velocity of phase 0, which determines the velocity of impulse conduction through the Purkinje system and ventricular myocardium. Hence the class I agents, which are sodium-channel blockers, interfere with the influx of sodium and reduces the velocity of phase 0 upstroke. This is reflected on the ECG as QRS complex widening. This effect on the QRS complex duration is more obvious with the class IC agents, which are more potent sodium-channel blockers. These agents produce this effect by blocking a receptor in the sodium channel during systole, interfering with the rapid influx of sodium. The drug dissociates during diastole. When the heart rate increased, there is less time for drug to dissociate, resulting in an increase in the number of receptors blocked, and hence a further decrease in sodium influx, a slowing of phase 0 upstroke and decrease in the conduction velocity. As a result there is a rate-related increase in QRS widening. This is termed use-dependent effect of the drug. Importantly, the abrupt increase in QRS width can be mistaken for ventricular complexes, especially when there is a long PR interval and the P wave merges with the T wave and is not obvious. These wide QRS complexes at a rapid rate may be confused with ventricular tachycardia. This was the concern with this patient. However, the occurrence of P waves before each QRS complex with a stable PR interval establishes a diagnosis of sinus tachycardia.

continues



ECG 30C Analysis: Sinus tachycardia, first-degree AV block (prolonged AV conduction), premature ventricular complex, intraventricular conduction delay (use dependent effect of antiarrhythmic drugs)

ECG 30C shows an ECG rhythm strip after the test was stopped. With continued ECG monitoring, the rate remains at 100 bpm. The QRS width is still wide at 0.20 second. However, the thirteenth QRS complex (^) is premature and has a wider duration and different morphology. This is a premature ventricular complex, after which there is a pause (↔). During the pause, a clear P wave (+) can be seen before the next QRS complex, with a PR interval of 0.26 second (⊐). Now that a PR interval can be established, it can be seen that there are indeed P waves before each of the QRS complexes, *ie*, the notching seen on the down slope of the T wave (↓). The interval from the P wave to QRS

(PR interval) is 0.26 second (⊐), identical to the PR interval after the premature ventricular complex and identical to the PR interval during normal sinus rhythm (ECG 30A). Therefore, this is a sinus rhythm with a rate-related increase in the QRS complex duration. When the baseline PR interval is prolonged, an increase in the heart rate will cause the P wave to merge with the T wave, and hence not be obvious. The identification of the P wave after the post-ectopic pause confirms the fact that this is a sinus tachycardia and hence the QRS widening is a result of use dependency. ■

Notes

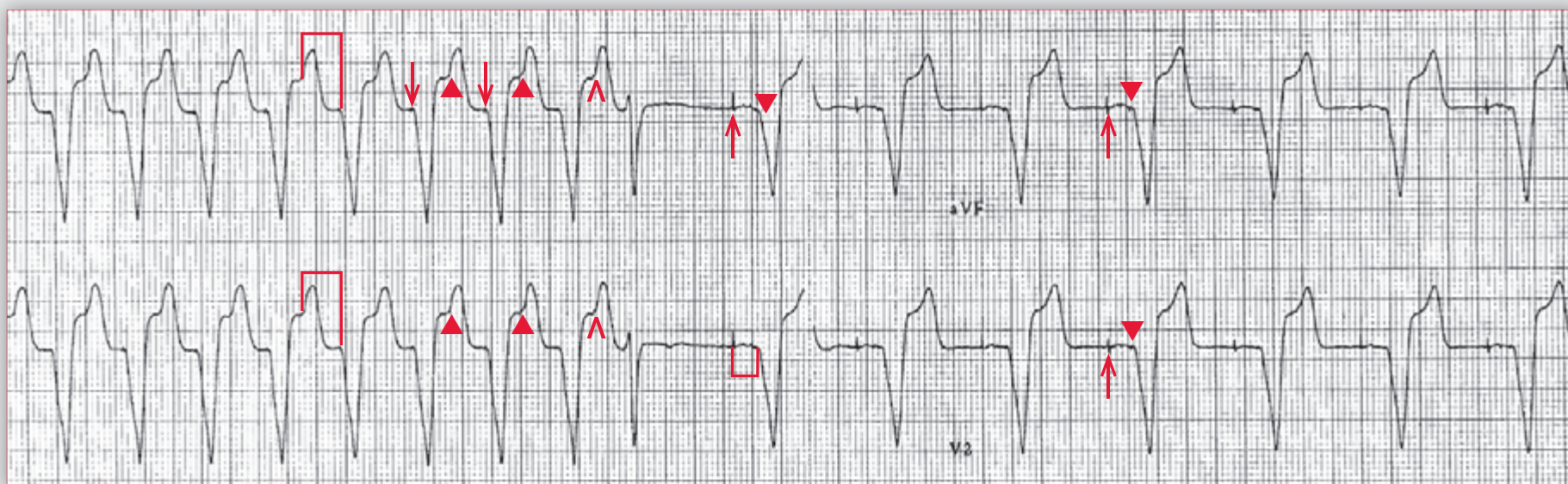
Since having a pacemaker placed for symptomatic bradycardia and Mobitz type II second-degree heart block, a 70-year-old female has been having bouts of distressing palpitations. Several emergency department visits have been unrevealing. She was admitted to the hospital for observation. This rhythm strip was recorded at night while she was asleep.

What appears to be the nature of her symptomatic tachycardia?

How would this tachycardia be treated?



Podrid's Real-World ECGs



ECG 31 Analysis: Pacemaker mediated tachycardia

There are two simultaneously recorded rhythm strips. The first 8 QRS complexes are regular at a rate of 126 bpm. The complexes are wide (duration = 0.16 sec). A very small pacemaker stimulus (↓) can be seen prior to each QRS complex. The ninth QRS complex is premature and narrower (duration = 0.12 sec). There is no P wave before this QRS complex and hence it is likely junctional. Following this premature complex there is a pause, after which there is a pacemaker stimulus preceding the P wave (↑) and also a pacemaker stimulus preceding the QRS complex (▼). Therefore, this is an AV sequentially paced rhythm at a rate of 72 bpm, which represents the lower rate limit of the pacemaker. The AV delay is 0.20 second (□). Although all the QRS complexes are paced and have the same morphology, it can be seen that the first 8 QRS complexes have a notching of the ST segment (▲) and a more peaked P wave that is not seen in the AV sequentially paced QRS complexes that follow the pause. These are P waves, and the associated PR interval is 0.20 second (□), identical to the AV delay of the pacemaker. The abrupt slowing of the rate following the premature junctional complex suggests that the initial rhythm was the result of a reentrant mechanism, which was terminated by the junctional

complex as it caused the AV node to be refractory. Importantly, the last QRS complex of the tachycardia still has a P wave (^), suggesting that the P wave is retrograde—the result of the paced QRS complex. Hence, this wide complex tachycardia is termed a pacemaker-mediated tachycardia, which may also be called an endless loop tachycardia. This occurs when there is a dual-chamber pacemaker and intact VA conduction through the intrinsic AV node. A dual-chamber pacemaker serves as a second AV pathway in addition to the normal AV node–His-Purkinje system. In this situation, a ventricular paced complex results in retrograde atrial activation. If this atrial impulse is sensed by the atrial lead, there will be a ventricular impulse generated and a paced ventricular complex. If VA conduction again occurs, a reentrant mechanism established involving the pacemaker and the intrinsic conduction system. Hence the pacemaker will continuously pace the ventricle at its upper rate limit, which is 126 bpm in this case. The premature junctional complex depolarizes the ventricle early, inhibiting the pacemaker and hence terminating the reentrant tachycardia. It also causes the AV node to be refractory and not capable of conducting any impulse retrogradely.

continues

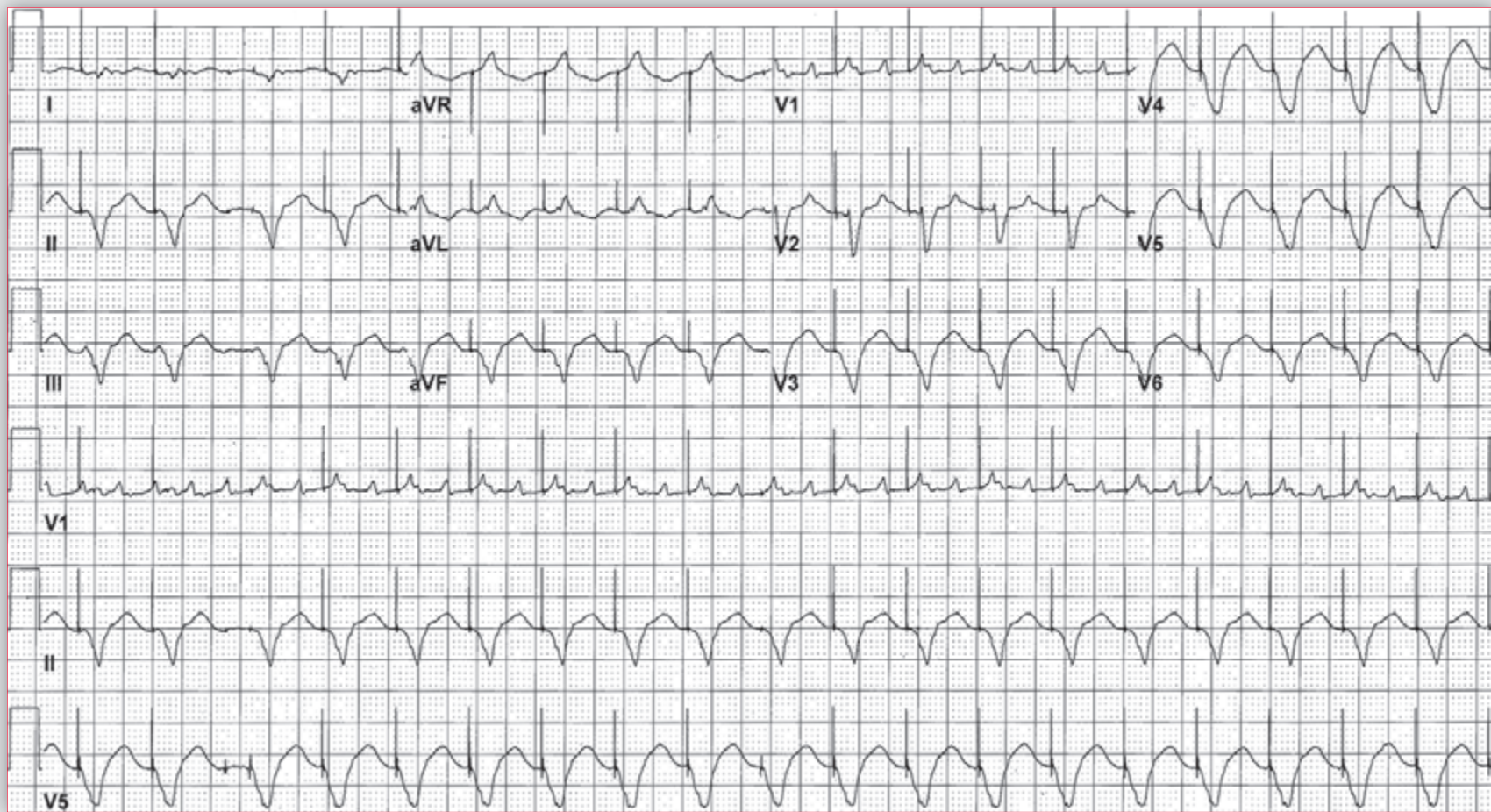
A pacemaker-mediated tachycardia can be terminated by deactivating atrial (and also ventricular) sensing, so that any retrograde atrial activity is not sensed by the atrial lead; hence there will not be a resultant ventricular impulse. This can be achieved with a magnet, which turns off all sensing, converting the pacemaker into a DOO mode, *ie*, a fixed-rate AV sequential pacemaker. In this situation, atrial and ventricular pacing stimuli will be at a constant at a fixed rate (*ie*, at the lower rate limit of the pacemaker). The stimuli will not capture if the atria and ventricles have been stimulated by native complexes and are therefore refractory. If the myocardium is capable of being stimulated, then the

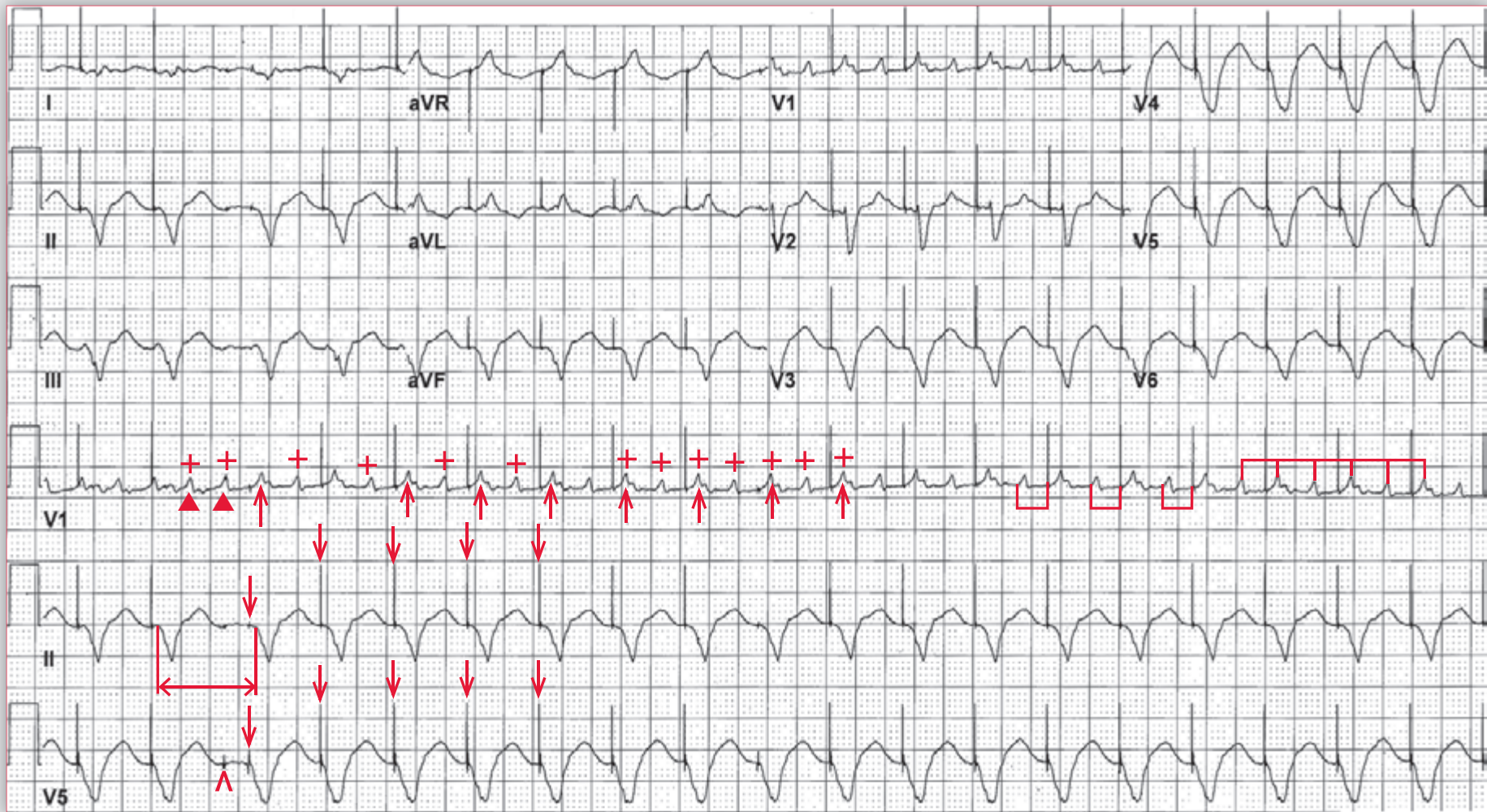
stimuli will result in paced complexes. This will appear as intermittent capture. For a permanent therapy, the PVARP (post-ventricular atrial refractory period) can be extended. This represents the time after a ventricular stimulus that the atrial lead is unable to sense any atrial activity. Known as the blanking period, the atrial lead will be “blind” to any atrial activity during this time and hence a retrograde P wave will not be sensed and a ventricular stimulus will not be delivered. The PVARP also determines the upper rate limit of the pacemaker, and by extending this parameter, the upper rate limit is reduced. ■

Two weeks after receiving a pacemaker for complete heart block, a 52-year-old man is undergoing a routine device interrogation. At his initial exam, he is noted to have a moderate tachycardia, and an ECG is obtained.

What is your interpretation of the ECG, and what is the cause of the tachycardia?

How can this be treated?





ECG 32 Analysis: Atrial flutter, AV sequential pacing and P wave synchronous ventricular pacing (or atrial activated ventricular pacing)

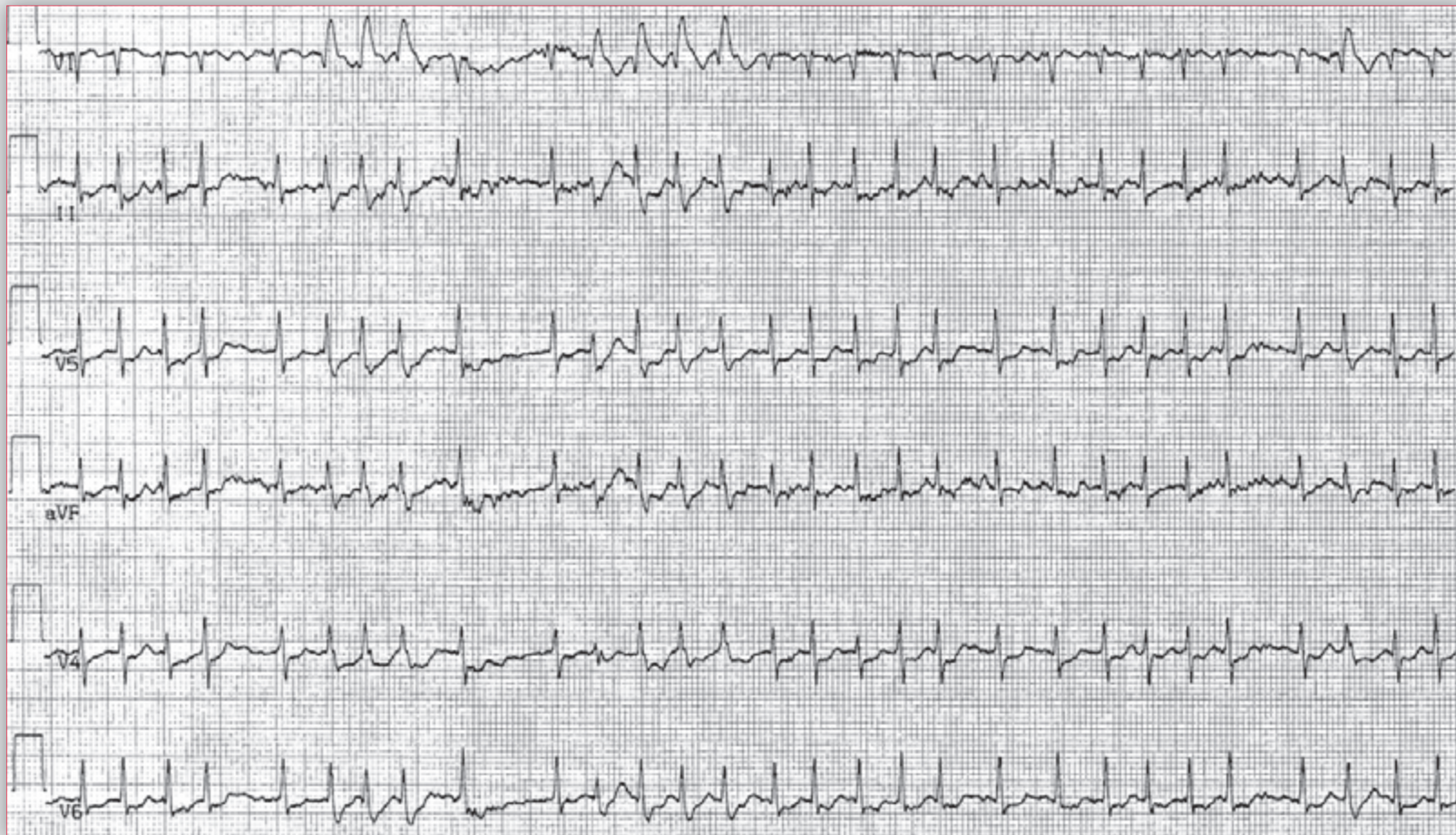
There is a regular rhythm at a rate of 130 bpm, although there is one long RR interval (\leftrightarrow). A single pacing stimulus (\downarrow) is seen before each QRS complex. Hence this represents ventricular pacing. As the pacemaker rate is 130 bpm, the pacemaker must be responding to atrial activity and functioning as a P-wave synchronous ventricular pacemaker or atrial-activated ventricular pacemaker. After the pause, there is a pacing stimulus seen before the P wave (\wedge) as well as before the QRS complex; this is AV sequential pacing, although there does not appear to be a P wave in response to the atrial stimulus. Therefore, the patient has a dual-chamber pacemaker. Although P waves are not seen in most leads, there is evidence of atrial activity seen in lead V1 ($+$). During the pause there are two atrial waveforms seen (\blacktriangle) at a rate of 260 bpm. Some of the atrial waveforms are superimposed on the end of the QRS complexes (\uparrow). However, whenever atrial waveforms are seen, they are on time (at a rate of 260 bpm), including the waveforms that are on the QRS complexes. The atrial rhythm is regular at a rate of 260 bpm (\square). Therefore, the atrial rhythm is atrial flutter. As the atrial rate is twice the ventricular paced rate, the pacemaker is sensing every other atrial flutter wave and is pacing at 130 bpm. The pacemaker is not capable of sensing each of the atrial flutter waves, and therefore there is 2:1 block. It can be seen that there is a fixed relationship between the atrial flutter wave and the pacing stimulus (0.20 sec) (\sqcup), which is the AV delay of the pacemaker. Therefore, this wide QRS complex is a pacemaker-related tachycardia, a result of the pacemaker tracking the atrial flutter with a pattern of 2:1 block, *ie*, only every other P wave is sensed, resulting in a ventricular paced complex.

This wide complex tachycardia is due to the pacemaker tracking an atrial arrhythmia. This can be treated with a magnet to disable all sensing and hence convert the pacemaker to a DOO mode, or a fixed-rate AV sequential pacemaker. The atrial activity will not be sensed by the atrial lead; hence there will be no resultant ventricular impulse. In this situation the patient's native rhythm will be seen and the ventricular response rate will be determined by AV nodal conduction. However, atrial and ventricular pacing stimuli will be at a constant or fixed rate (*ie*, at the lower rate limit of the pacemaker). The stimuli will not capture if the atria and ventricles have been stimulated by native complexes and are therefore refractory. If the myocardium is capable of being stimulated, then the stimuli will result in paced complexes. This will appear as intermittent capture. Most pacemakers have a programmable feature that allows for "mode switching." In this situation, if there is a rapid atrial rate, the pacemaker automatically switches to VVI pacing, *ie*, it becomes a demand ventricular pacemaker, and there is no longer any atrial sensing or atrial pacing. The ventricular rate will be determined by conduction through the AV node, with only ventricular pacing, which will occur whenever the intrinsic rate falls below the lower rate limit of the pacemaker. ■

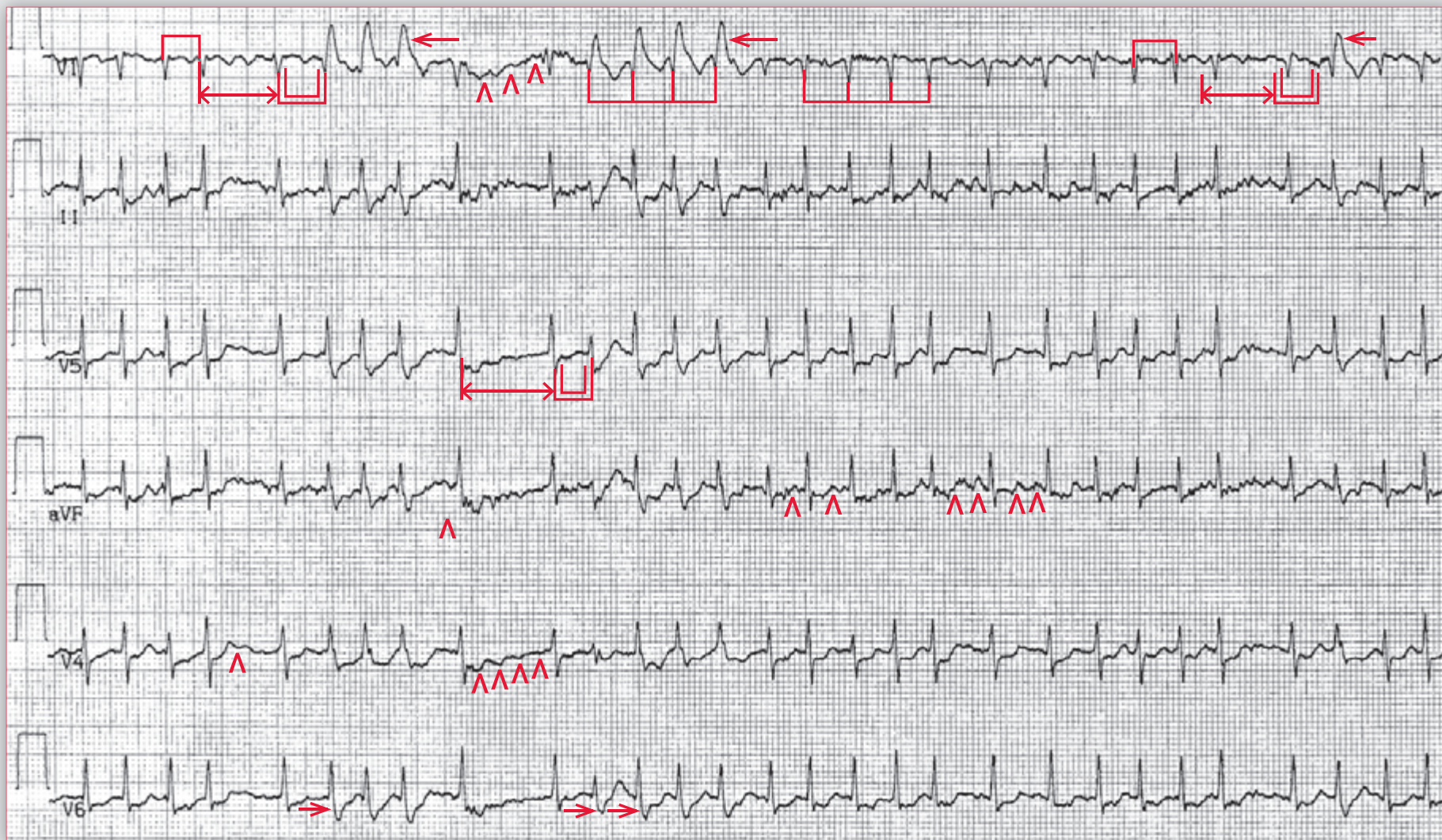
Notes

A 68-year-old man presents to the emergency department because of palpitations that began several hours before presentation. He has no known heart disease, although he does have COPD and has recently been using his inhalers more frequently because of a COPD exacerbation. Although he does not have any other symptoms, he is bothered by the palpitations because they have persisted even though he has withheld any further inhaler therapy.

What is your interpretation of the presented ECG, and what is the name of the form of aberrancy depicted?



Podrid's Real-World ECGs



ECG 33 Analysis: Atrial fibrillation, Ashman phenomenon

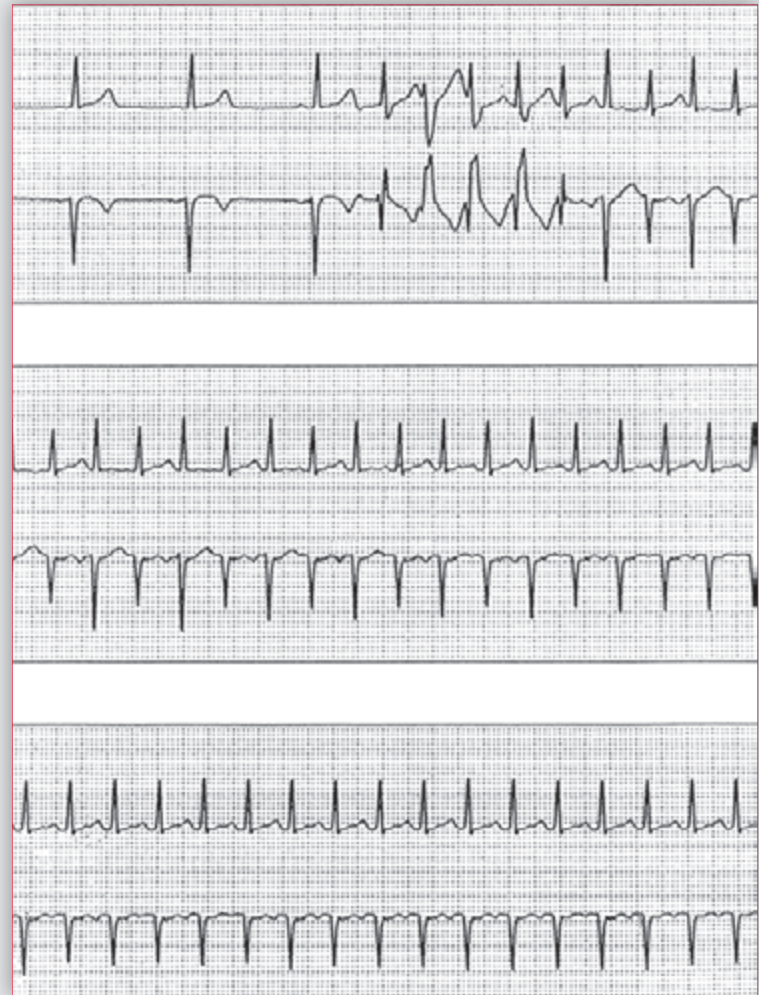
The ECG shows six simultaneously recorded rhythm strips. There is an irregularly irregular rhythm at a rate of 174 bpm. There are only three supraventricular rhythms that are irregularly irregular, including sinus arrhythmia (one P-wave morphology and stable PR interval); multifocal atrial rhythm (wandering atrial pacemaker) if the rate is < 100 bpm or multifocal atrial tachycardia if the rate > 100 bpm (> 3 P-wave morphologies and different PR intervals); or atrial fibrillation (no organized P waves). Atrial flutter or atrial tachycardia may be irregular, but there is a pattern to the irregularity, based on the degree of AV nodal blockade or conduction (*ie*, 2:1, 3:1, 4:1 or variable). These rhythms may be regularly irregular. There are no clear or organized P waves seen before or after any QRS complex. There are rapid and irregular undulations of the baseline, most evident in leads V1, V4, and aVF (Λ). Hence this is atrial fibrillation. The QRS complexes manifest two different widths. The narrower complexes have a duration of 0.08 second, and they have a normal morphology. The wide complexes (0.14 sec) have a typical right bundle branch block morphology with a RSR' complex in lead V1 (←) and a broad S wave in leads V4–V6 (→). The wide QRS complexes are thus aberrant. This is not a typical rate-related aberrancy, as the RR intervals of the wider QRS complexes are not different from those that are associated with some of the narrow QRS complexes (□). Indeed, there are occasional RR intervals that are shorter (◻) but that are not associated with aberrancy. There is a long-short RR interval (↔, ◻◻) noted before the wider QRS complexes. Thus, the aberrancy is a result of an Ashman phenomenon: aberration is related to abrupt change in heart rate, *ie*, slower rate (long RR interval) followed by a faster heart rate (shorter RR interval).

The usual form of aberration, which is rate-related and associated with faster heart rate (functional bundle branch block), is due to conduction block resulting from underlying conduction system disease (and the inability of the conduction system to conduct with a faster rate). In contrast, the Ashman phenomenon is the result of normal physiologic changes in refractoriness of the His-Purkinje system. At slower heart rates (longer RR interval) His-Purkinje refractoriness increases, while with faster heart rates (shorter RR interval), refractoriness decreases. Whenever there is abrupt change in rate, *ie*, slower to faster, refractoriness does not have time to shorten or adapt; hence there is block of conduction due to the long refractoriness, resulting in aberration. This is more likely to be seen in atrial fibrillation, which is associated with frequent, marked irregularity in RR intervals and more frequent episodes of long-short RR intervals. The Ashman phenomenon can be seen whenever there is an abrupt change in heart rate, going from slow to faster. The Ashman phenomenon is most often associated with a right bundle branch aberration, likely because the right bundle refractory period is slightly longer than that of the left bundle. The Ashman phenomenon may last for more than one complex. As can be seen, there is a three- and four-beat run of aberrant QRS complexes as well as a single aberrant complex. The presumed mechanism for this is that with the right bundle branch and conduction only via the left bundle, there is the potential for retrograde conduction through the right bundle (concealed penetration), causing it to remain refractory to antegrade conduction. ■

Core Case 34

Presented are the rhythm strips from ambulatory monitoring of a patient with an arrhythmia.

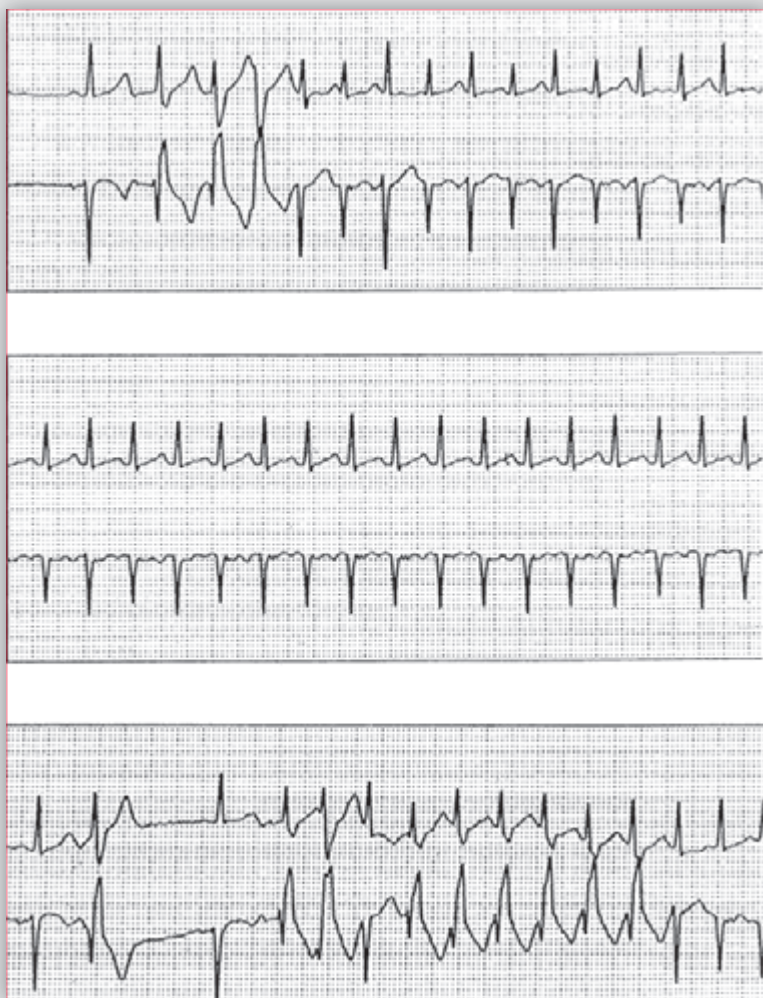
ECG 34A



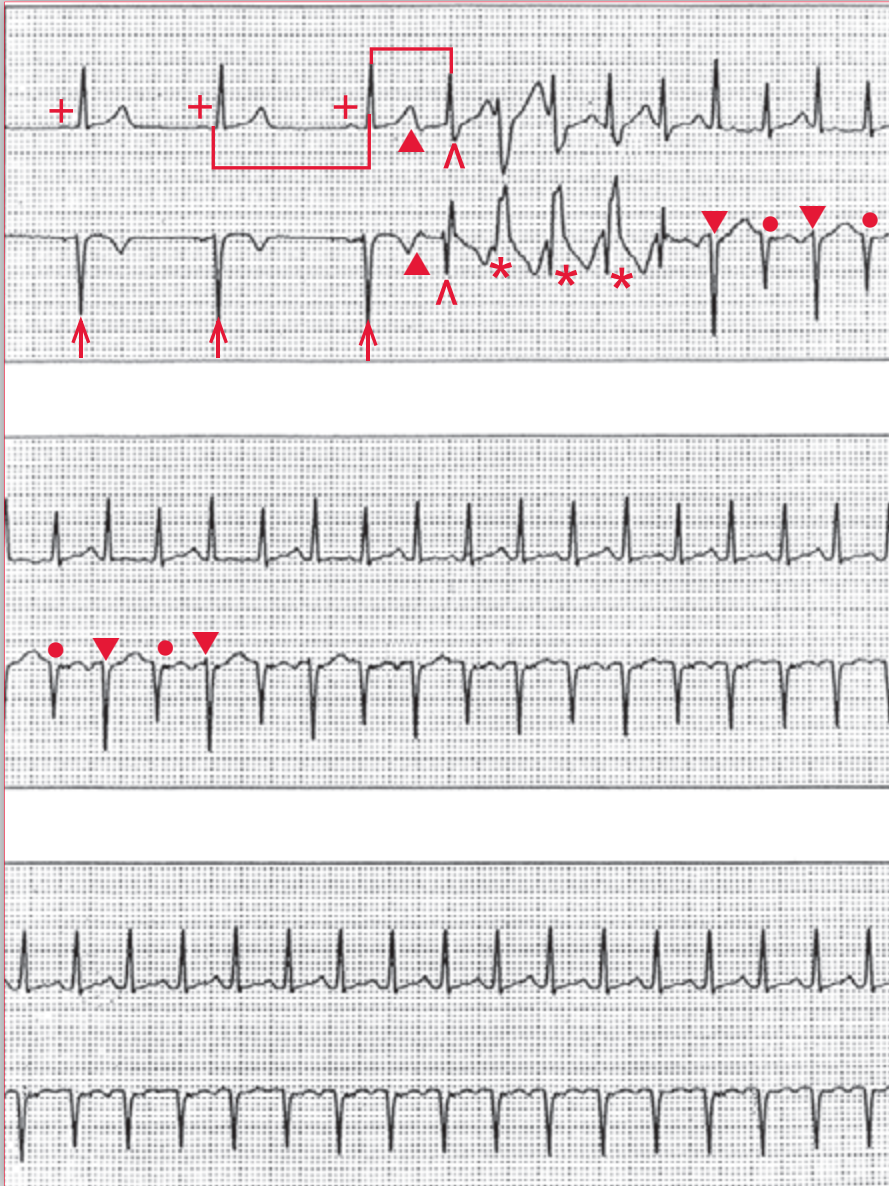
What is your interpretation of the QRS complexes over the course of the strips (tracings A and B)?

What aberrant phenomena are depicted?

ECG 34B



Podrid's Real-World ECGs



ECG 34A (and 34B) Analysis: Normal sinus rhythm, premature atrial complex, atrioventricular nodal reentrant tachycardia, QRS (electrical) and T-wave alternans, Ashman phenomenon

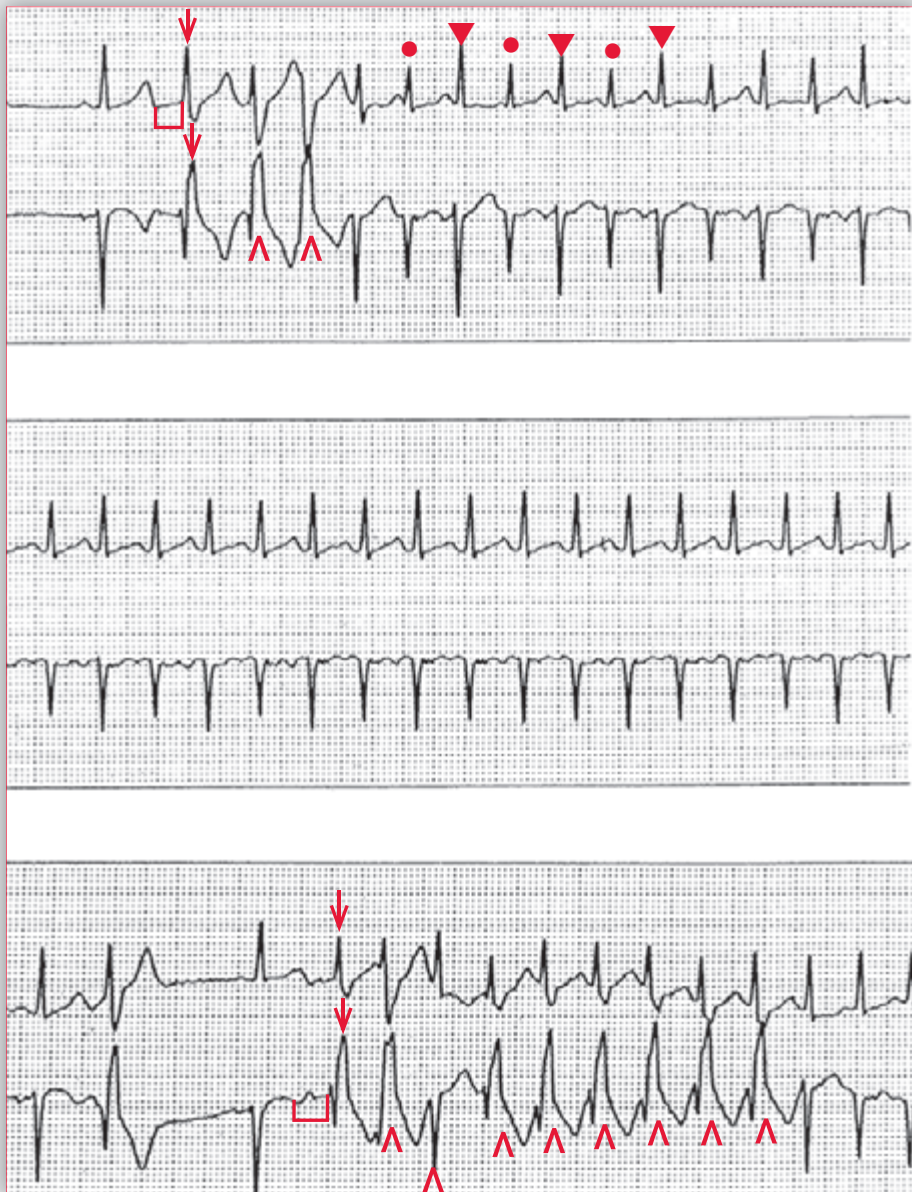
ECG 34A shows that there are three rhythm strips, obtained from an ambulatory monitor, that are continuous. The first three QRS complexes (†) in the top rhythm strip are narrow (duration 0.08 sec), and they are preceded by a P wave (+) with a stable PR interval (0.14 sec). The fourth QRS complex is premature (^) and there is a P wave (▲) seen before this premature QRS complex with a PR interval of 0.20 second; the P wave is negative. This is a premature atrial complex. Following this premature complex are three wide QRS complexes (*) (duration = 0.16 sec) with a right bundle branch block morphology; the rate is 180 bpm. Thereafter, all the QRS complexes are narrow (0.08 sec) with a morphology that is identical to the initial sinus complexes (†); they are at a regular rate of 180 bpm. Hence the initial aberrated complexes (^,*) do not have rate-related aberration, as they are at the same rate as the narrow QRS complexes that follow. They are, however, associated with a long-short RR interval (⊐,⊑), *ie*, a slower rate with the abrupt increase in rate at the onset of the tachycardia. The aberrant QRS complex are, however, due to an Ashman phenomenon. The Ashman phenomenon can occur with the abrupt onset of any rapid supraventricular tachycardia (abrupt slow to fast heart rate) and may be seen with one or several QRS complexes.

The Ashman phenomenon is the result of normal physiologic changes in refractoriness of the His-Purkinje system. At slower heart rates (longer RR interval) His-Purkinje refractoriness increases, while with faster heart rates (shorter RR interval) refractoriness decreases. Whenever there is abrupt change in rate, *ie*, slower to faster, refractoriness does not have time to shorten or adapt; hence there is block of conduction due to the long refractoriness, resulting in aberration. The Ashman

phenomenon can be seen whenever there is an abrupt change in heart rate, going from slow to faster. The Ashman phenomenon is primarily associated with a right bundle branch aberration, likely because the right bundle refractory period is slightly longer than that of the left bundle. The Ashman phenomenon may last for more than one complex. As can be seen, there is a three- and four-beat run of aberrant QRS complexes as well as a single aberrant complex. The presumed mechanism for this is that with the right bundle branch and conduction only via the left bundle, there is the potential for retrograde conduction through the right bundle (concealed penetration), causing it to remain refractory to antegrade conduction.

This narrow complex tachycardia is initiated by a premature atrial beat with a PR interval that is longer than that of the sinus complexes. There are no P waves seen with this tachycardia. Hence this is a no-RP tachycardia, and the most common etiology for this is an atrioventricular nodal reentrant tachycardia. Also observed is QRS (electrical) and T-wave alternans (●,▼), with beat-to-beat changes in QRS and T wave amplitude. QRS and T-wave alternans is often seen with any rapid supraventricular tachycardia and is due to beat-to-beat changes in calcium influxes. The alternans does resolve and is not seen during the tachycardia in the bottom rhythm strip. Electric alternans can also be seen with an acute myocardial infarction, decompensated heart failure, and a dilated cardiomyopathy in these conditions it is also the result of changes in calcium influxes. Alternans is also seen with tamponade, in which case the mechanism is mechanical, with a swinging of the heart within the fluid-filled pericardial sac (pendulum effect). In this situation, there may also be P-wave alternans. *continues*

Podrid's Real-World ECGs



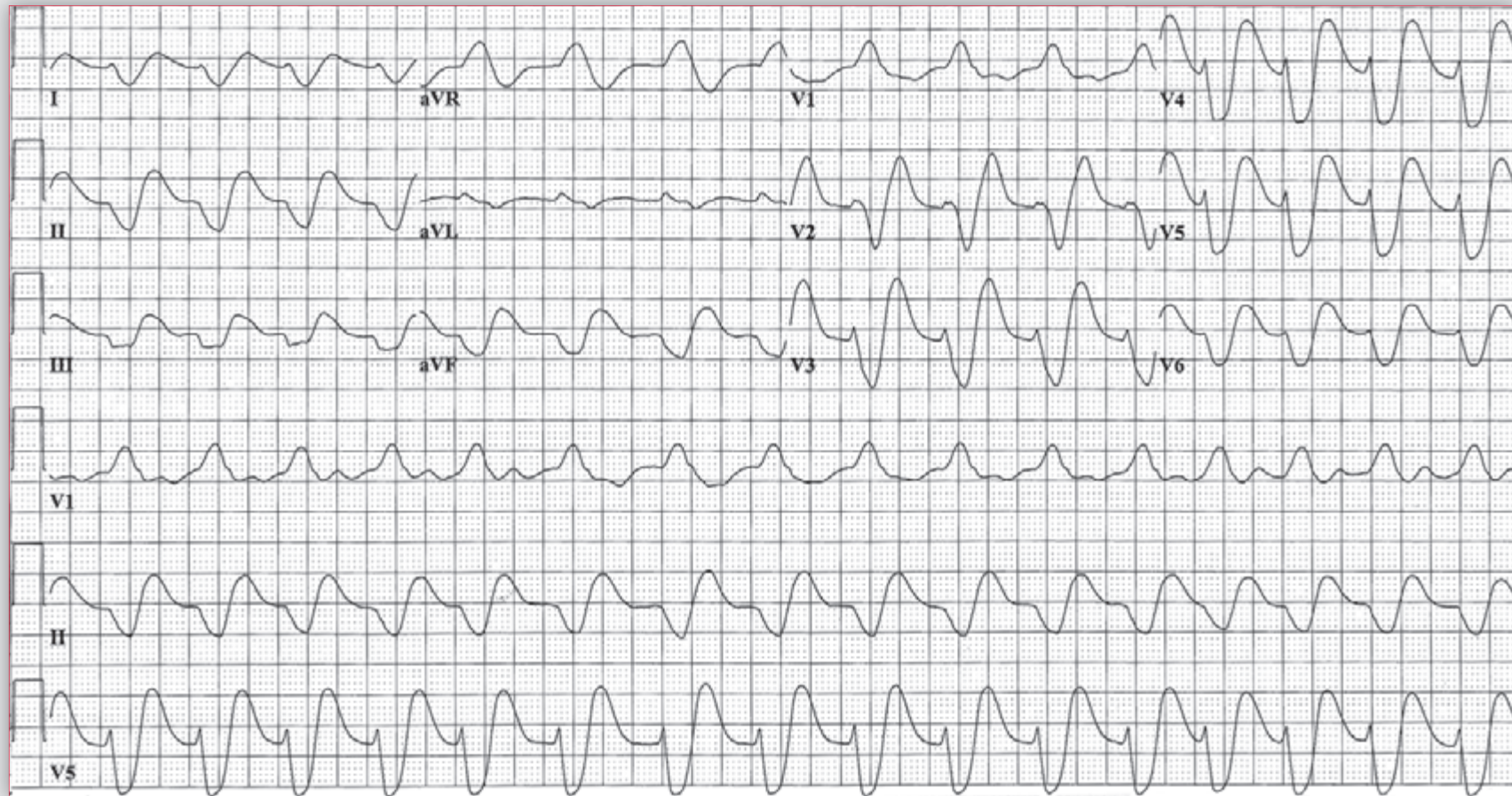
ECG 34B (and 34A) Analysis: Normal sinus rhythm, premature atrial complex, atrioventricular nodal reentrant tachycardia, QRS (electrical) and T-wave alternans, Ashman phenomenon

ECG 34B shows a series of rhythm strips from the same patient as ECG 34A and shows two more examples of the initiation of an atrioventricular nodal reentrant tachycardia. There are normal sinus complexes with a PR interval of 0.14 second followed by a premature atrial complex (↓) that has PR interval (⌊) longer (0.20 sec) than the sinus PR interval. The premature atrial complex initiates the arrhythmia. The premature atrial complex as well as the first few complexes of the AVNRT have a right bundle branch block morphology (^), a result of the Ashman phenomenon (*ie*, there is a long-short cycle before the aberrated QRS complex). As with ECG 34A, the beginning of the AVNRT is associated with QRS and T-wave alternans (●,▼). ■

Core Case 35

A 44-year-old diabetic man is brought via EMS to his local hospital, having been found unconscious at home. His initial triage includes assessment of vital signs are notable for hypotension, but normal heart rate. Laboratory data indicate that he has a hyperosmotic hyperglycemic

ECG 35A



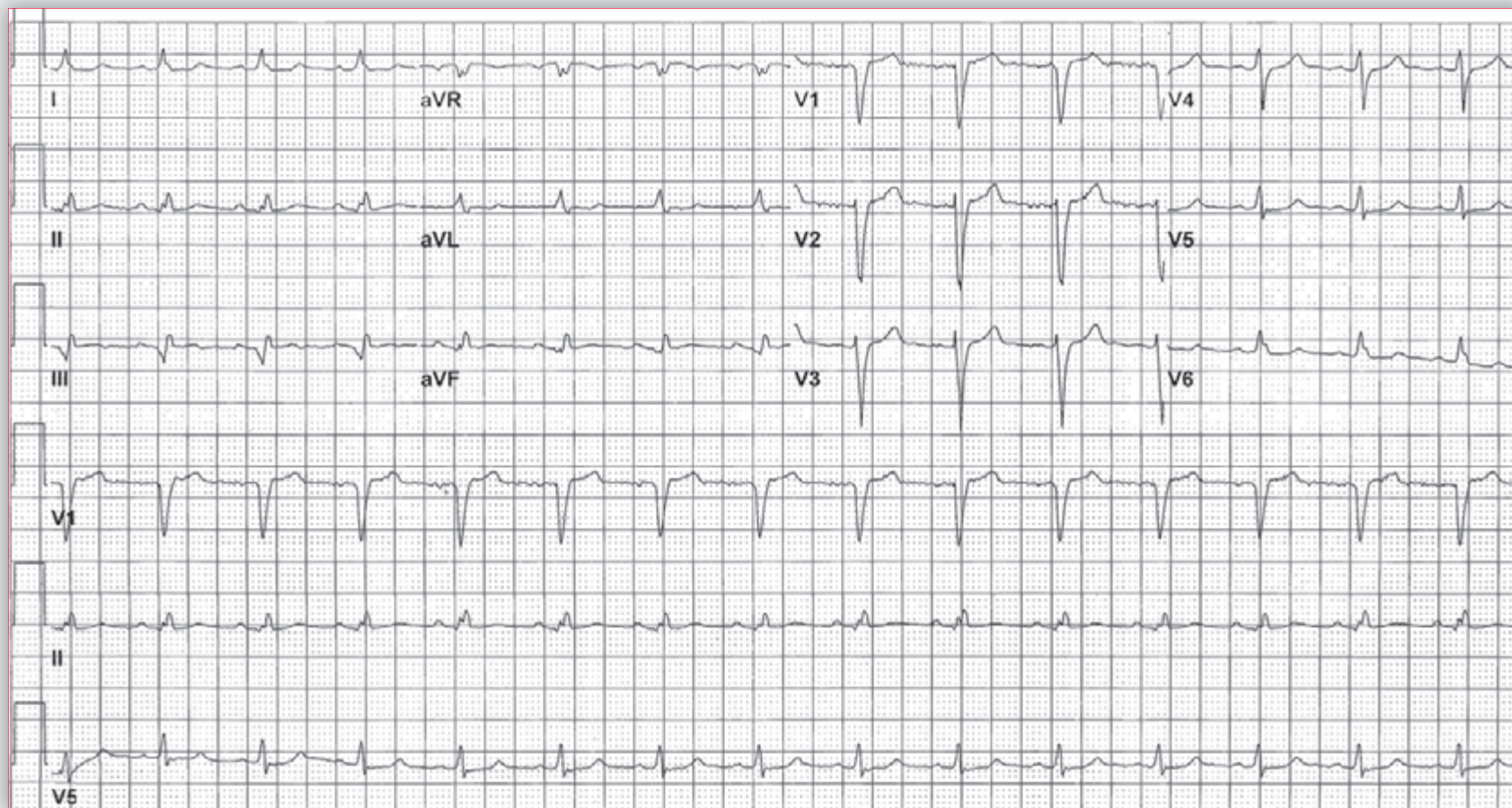
state accounting for the coma (glucose 800 mg/dL, serum osmolarity 400 mOsm/kg, pH 7.35 and no anion gap, and serum K 8.2 mmol/L). An ECG is performed (ECG 35A).

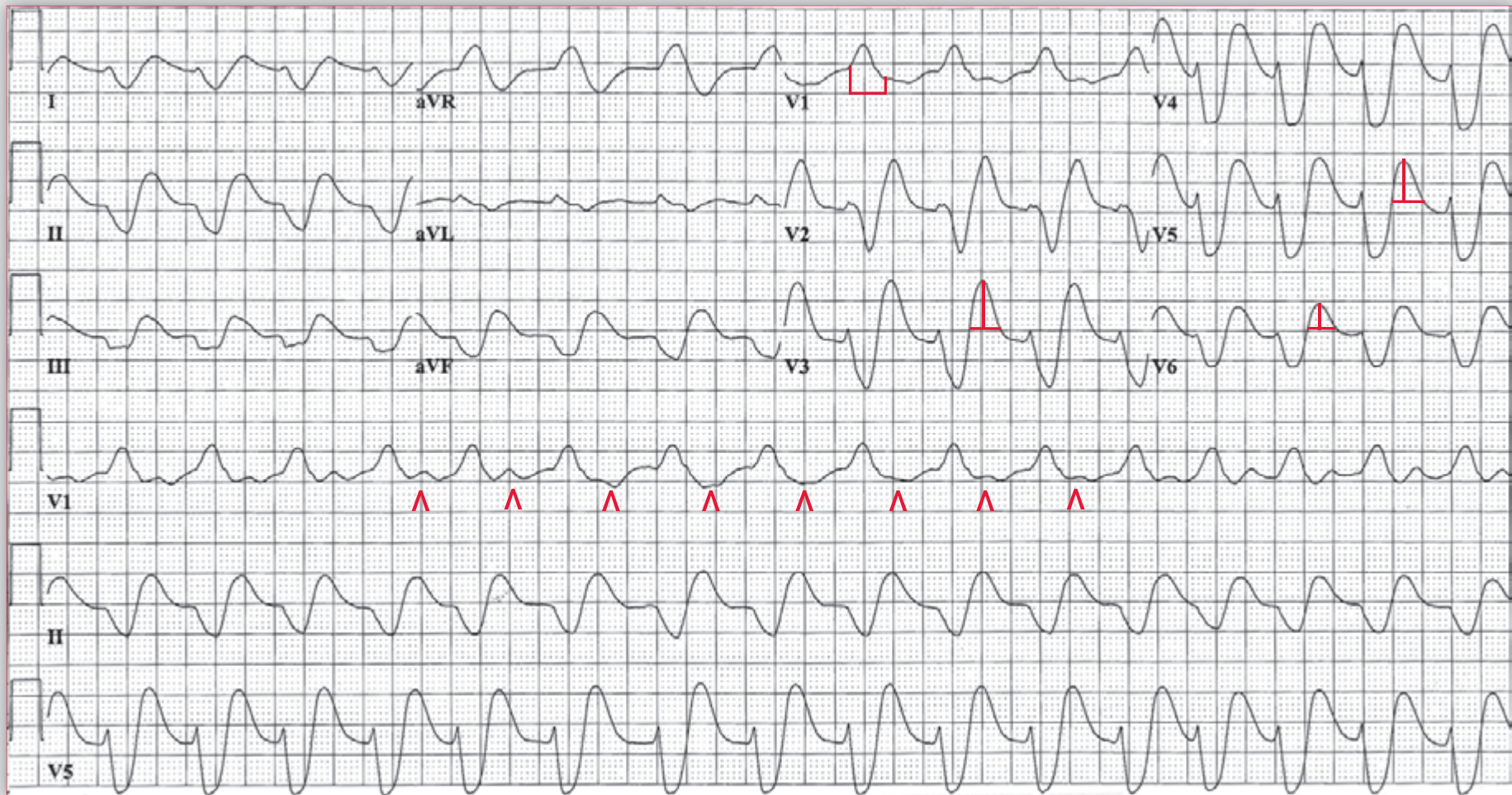
The patient is treated appropriately and the following day, a repeat ECG is obtained (ECG 35B).

What is the cause of the arrhythmic abnormalities depicted (ECG 35A)?

What is your interpretation of the second tracing (ECG 35B) in relation to the first tracing (ECG 35A)?

ECG 35B





ECG 35A Analysis: Wide complex rhythm, hyperkalemia

ECG 35A shows a regular rhythm at a rate of 90 bpm. There are no P waves seen in any of the leads. The QRS complex is very wide (0.24 sec), best established by measuring the QRS complex duration in lead V1 where the beginning and end of the QRS complex (J point) can be seen (\sqcup). The only condition associated with a QRS complex that is ≥ 0.24 second is hyperkalemia. In general a bundle branch block or a ventricular complex will not cause a QRS complex to be this wide. A very wide QRS complex (up to 0.22 sec) may be seen with a severe dilated cardiomyopathy and this is a result of diffuse fibrosis and slowing of impulse conduction. In addition, the T waves are symmetric (upstroke and downstroke equal) (\perp), which further supports hyperkalemia as the etiology. The QRS complex axis is indeterminate between -90° and $+/-180^\circ$. A wide QRS complex associated with an indeterminate axis occurs whenever there is direct stimulation of the ventricular myocardium and not when there is ventricular activation via the normal His-Purkinje system. This includes ventricular complexes, paced complexes or QRS complexes with a Wolff-Parkinson-White (WPW) pattern. In addition, there are irregularities of the ST-T waves (\wedge), best seen in lead V1. This finding, along with the indeterminate axis, suggests a diagnosis of ventricular tachycardia, along with hyperkalemia. This diagnosis is difficult to establish as the shift in the axis could be the result of the hyperkalemia.

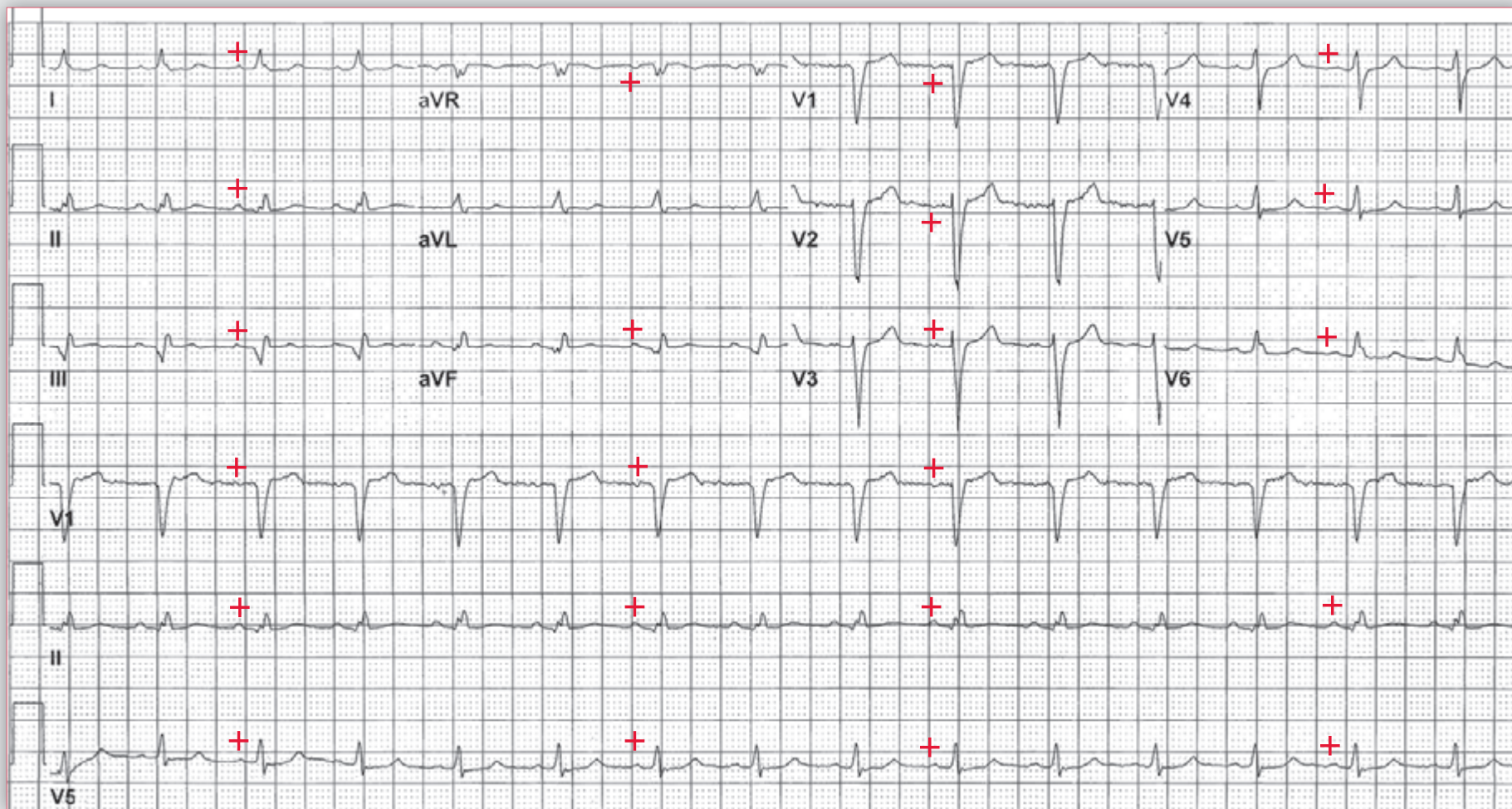
The QRS complex widening in hyperkalemia is related to the marked slowing of the upstroke velocity of phase 0 of the fast action potential.

The resting membrane potential of the fast action potential is -90 mV, and this is related to the balance between intra- and extracellular potassium concentrations. Intracellular potassium concentrations are far greater than extracellular concentrations and the balance is maintained by an energy-dependent Na^+/K^+ ATPase pump that exchanges sodium for potassium. The velocity of phase 0 upstroke determines the impulse conduction velocity through the Purkinje system and myocardial tissue and is due to rapid influx of sodium. The upstroke velocity is determined by the balance between the resting membrane potential and the threshold potential which is -60 mV. The closer the resting membrane potential is to the threshold potential, *ie*, if the resting membrane potential becomes less negative, the slower is the rate of sodium ion influx and the slower is the upstroke of phase 0. With hyperkalemia, there is a reduction in the balance between intra- and extracellular potassium, and the membrane becomes less negative. Therefore, the velocity of impulse conduction decreases, resulting in QRS complex widening. If the resting membrane potential is at the threshold potential of -60 mV, the membrane is inexcitable and asystole occurs.

The atrial myocardium is more sensitive to hyperkalemia and hence atrial asystole develops at lower potassium level, often before there is any substantial changes in QRS width. In this situation, there is still a sinus rhythm, but there is no atrial activation and hence no P wave or atrial asystole. This has been referred to a sinoventricular rhythm.

continues

Podrid's Real-World ECGs



ECG 35B Analysis: Normal sinus rhythm, low limb lead voltage

ECG 35B is from the same patient as ECG 35A and was obtained on the following day, after therapy for hyperkalemia was given and the potassium level corrected.

There is a regular rhythm at a rate of 90 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.22 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm with a first-degree AV block. The QRS complex duration is normal (0.08 sec) as is the morphology. The axis is normal between 0° and $+90^{\circ}$ (QRS complex positive in leads I and aVF. There

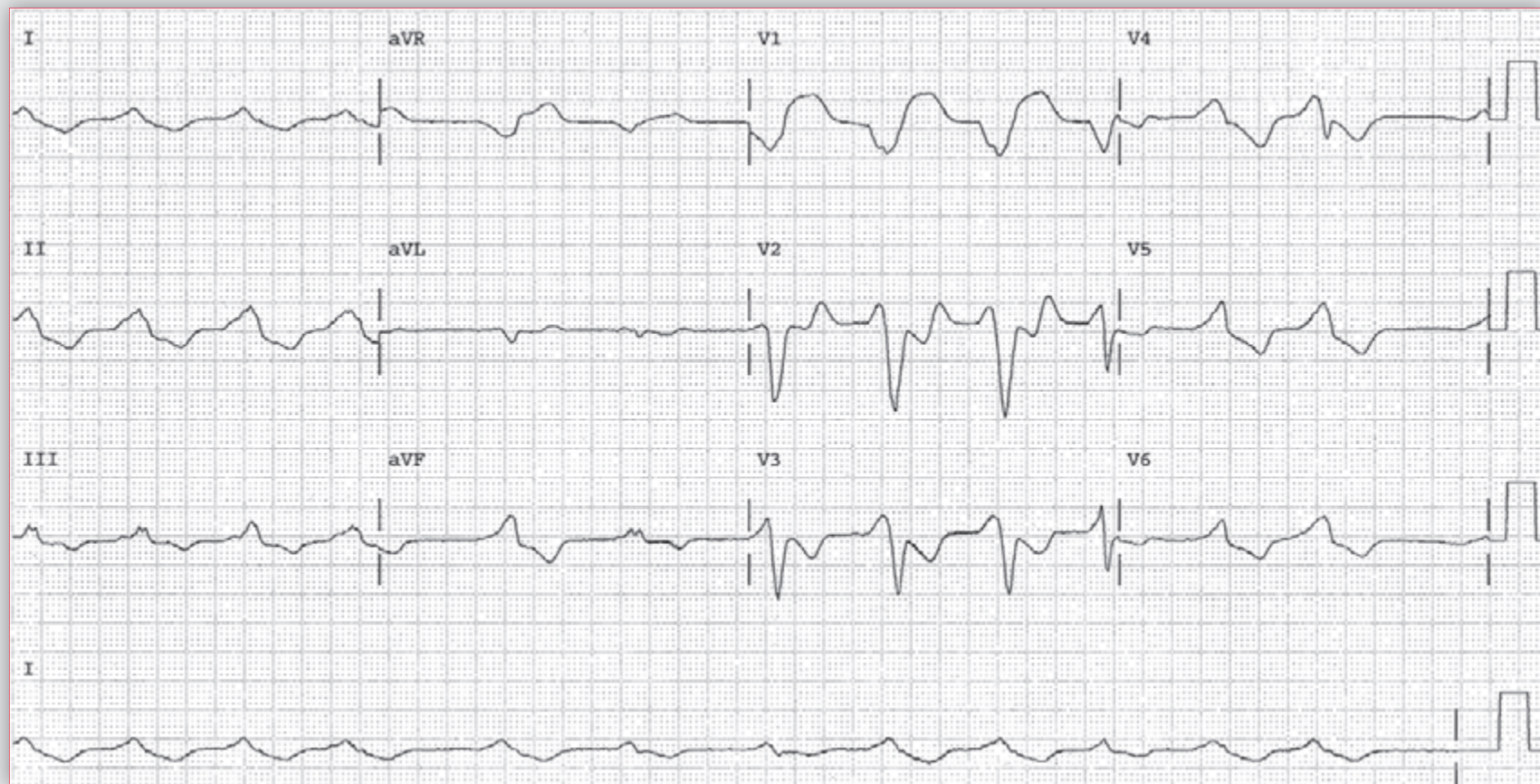
is low QRS complex voltage in the limb leads (*ie*, less than 5 mm in each limb leads). The QT/QTc intervals are normal (340/420 msec).

Acute therapy for hyperkalemia includes insulin (which forces potassium into the cells) and glucose (to prevent insulin-induced hypoglycemia). It has been recommended that calcium also be given to “stabilize the membrane” and enhance membrane excitability; however, how this happens is not understood, nor is it clear that this therapy is necessary. In addition, a resin (Kayexelate) was given to remove potassium. ■

Core Case 36

A 70-year-old man with a history of a non-ischemic cardiomyopathy and heart failure is receiving therapy with a β -blocker, ACE inhibitor, Lasix, and spironolactone. He has been well compensated, but recently developed an upper respiratory infection. Shortness of breath worsened, and he noted the onset of orthopnea and paroxysmal nocturnal dyspnea. Several days later, he noted profound weakness,

ECG 36A

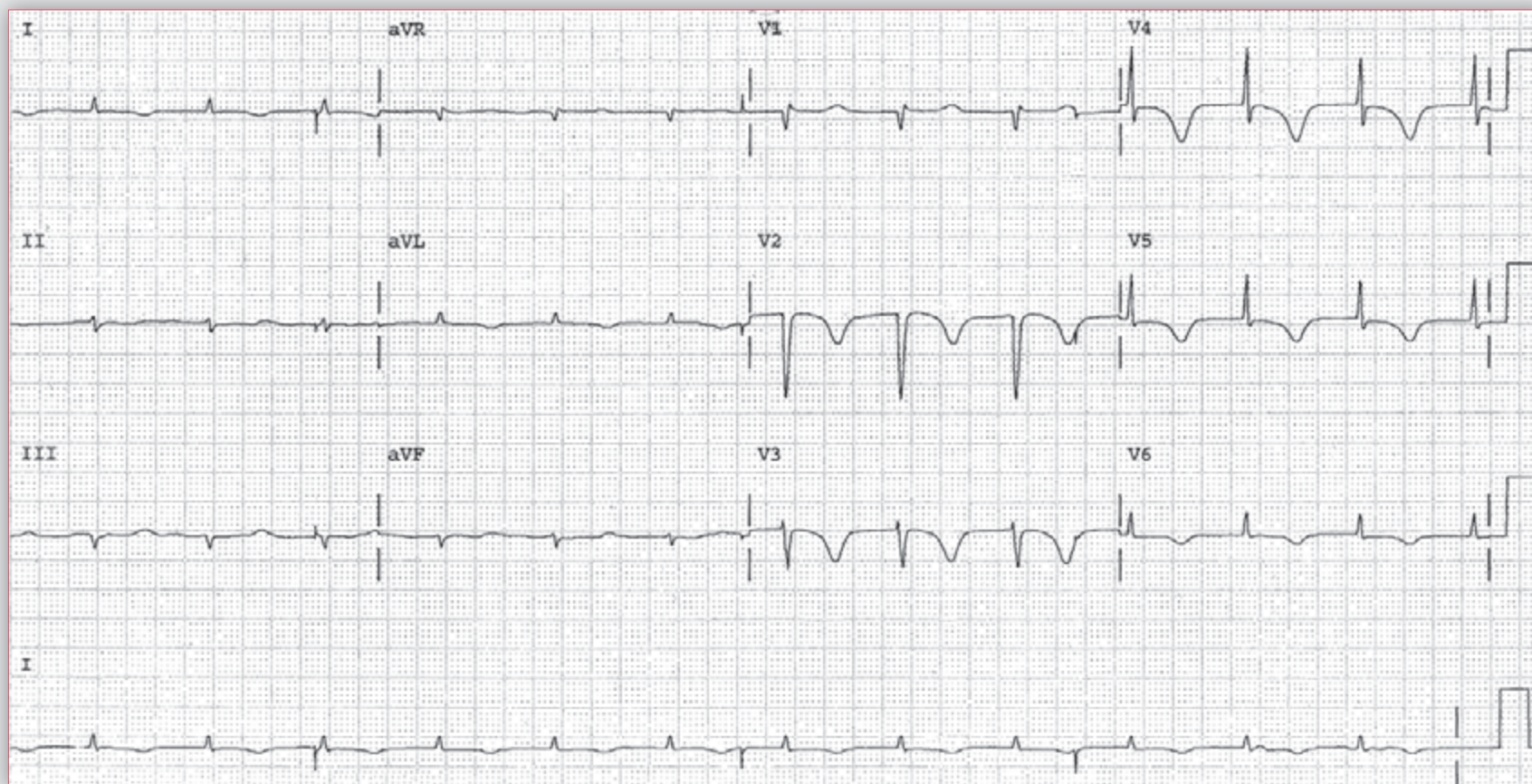


nausea, vomiting, and a reduction in urine output. Finally, he went to an emergency department where a physical examination and chest x-ray were consistent with pulmonary edema. Laboratory data revealed a serum creatinine of 4.5 (baseline 1.8) and a BUN of 146. Serum potassium was 7.6 mmol/L. An ECG is obtained (ECG 36A). He is treated with IV Lasix and a renal consult is called for consideration of dialysis.

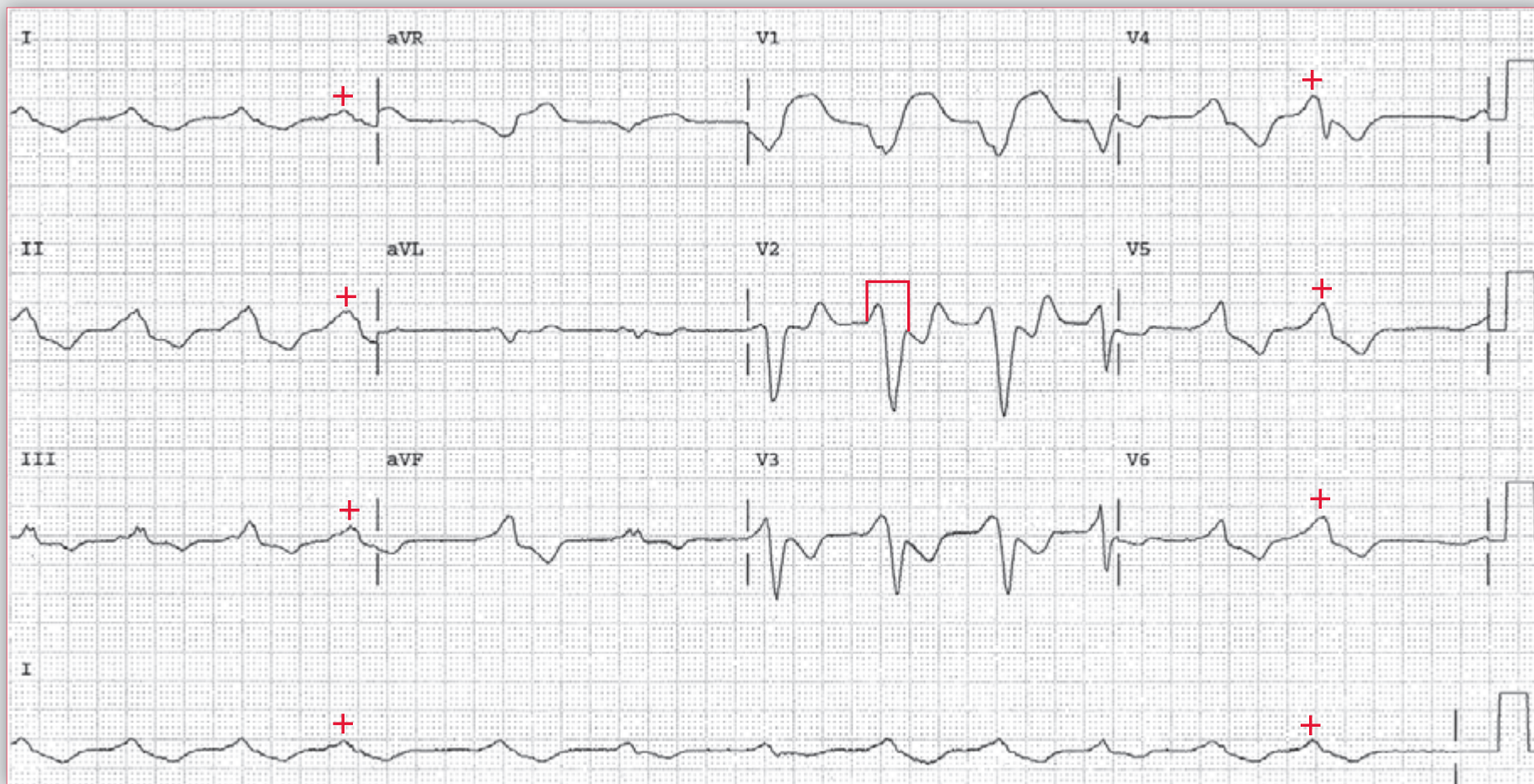
What is the abnormality noted on the ECG, and what therapy is indicated?

What is the cause for the patient's clinical presentation?

ECG 36B



Podrid's Real-World ECGs



ECG 36A Analysis: Wide complex rhythm due to hyperkalemia, premature complexes, diffuse ST-T wave abnormalities

ECG 36A shows a regular rhythm at a rate of 76 bpm. There are no P waves seen before or after any of the QRS complexes. The QRS complex duration is very prolonged (0.30 sec) (\square). The only situation in which the QRS complex duration is increased to ≥ 0.24 second is hyperkalemia. There are two complexes that are premature (the fourth and twelfth complexes [+]). Although these complexes have the same morphology, they are wider (0.32 sec). This is due to further slowing of conduction velocity when the QRS interval is shorter (or the ventricular rate is faster). It is not clear if these premature complexes are supraventricular or ventricular, although the fact that they have the same morphology as the other QRS complexes suggests that they are supraventricular in origin.

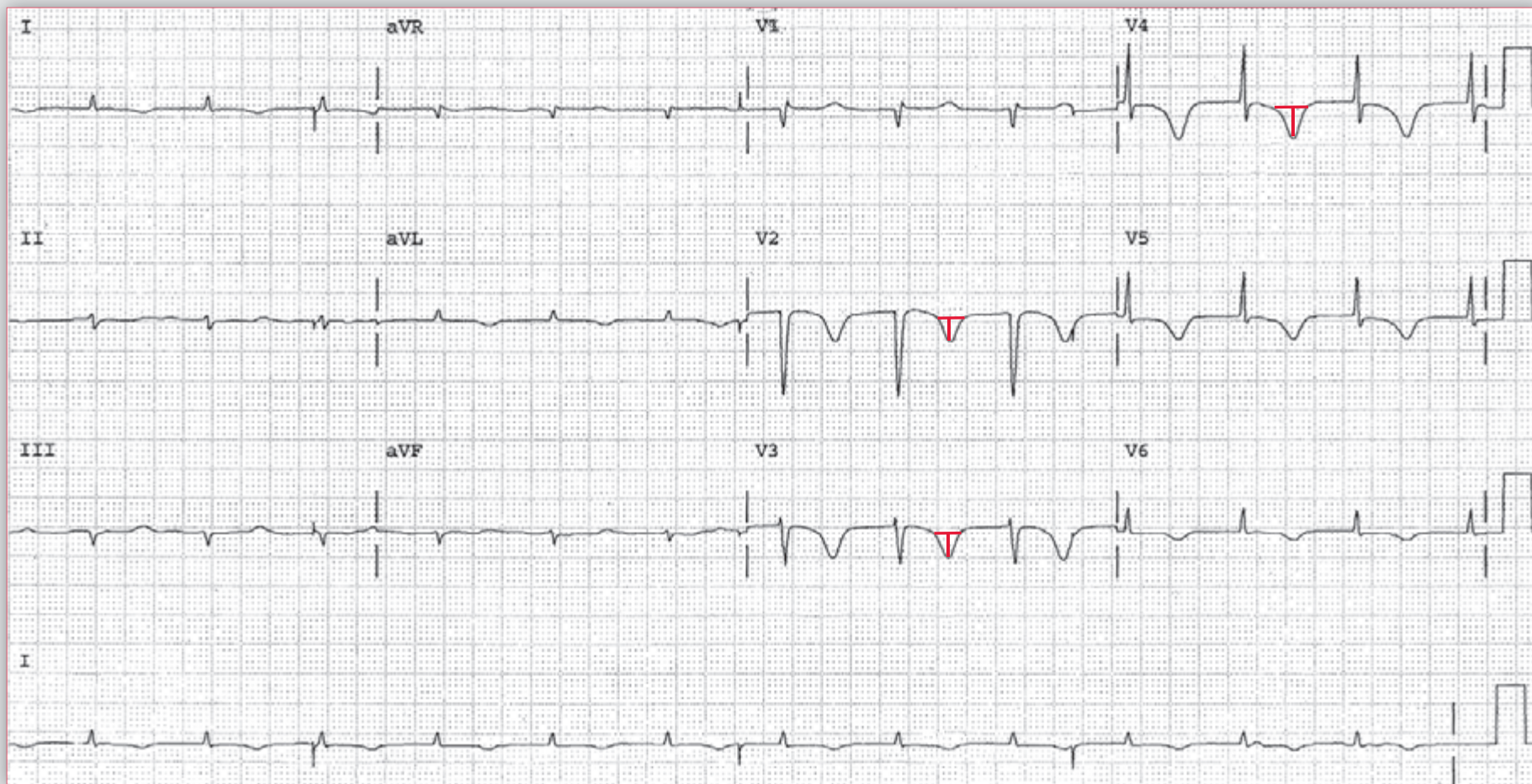
The QRS complex widening in hyperkalemia is related to the marked slowing of the upstroke velocity of phase 0 of the fast action potential. The resting membrane potential of the fast action potential is -90 mV, and this is related to the balance between intra- and extracellular potassium concentrations. Intracellular potassium concentrations are far greater than extracellular concentrations, and the balance is maintained by an energy-dependent Na^+/K^+ ATPase pump that exchanges

sodium for potassium. The velocity of phase 0 upstroke, which determines the impulse conduction velocity through the Purkinje system and myocardial tissue, is due to rapid influx of sodium. The upstroke velocity is determined by the balance between the resting membrane potential and the threshold potential which is -60 mV. The closer the resting membrane potential is to the threshold potential, *ie*, if the resting membrane potential becomes less negative, the rate of sodium ion influx is slower and so is the upstroke of phase 0. With hyperkalemia, there is a reduction in the balance between intra- and extracellular potassium, and the membrane becomes less negative. Therefore, the velocity of impulse conduction decreases, resulting in QRS complex widening. If the resting membrane potential is at the threshold potential of -60 mV, the membrane is inexcitable and asystole occurs.

The atrial myocardium is more sensitive to hyperkalemia and hence atrial asystole develops at lower potassium level, often before there is any substantial changes in QRS width. In this situation, there is still a sinus rhythm, but there is no atrial activation and hence no P wave or atrial asystole. This has been referred to a sinoventricular rhythm.

continues

Podrid's Real-World ECGs



ECG 36B Analysis: Junctional rhythm vs. sinoventricular rhythm, low-voltage limb leads, diffuse nonspecific ST-T wave abnormalities, left anterior fascicular block, hyperacute T waves

ECG 36B is from the same patient as ECG 36A and it was obtained 2 hours after therapy for hyperkalemia was given. There is a regular rhythm at a rate of 76 bpm. There are no P waves seen before or after any of the QRS complexes. The QRS complexes have a normal duration (0.08 sec) and they have a normal morphology, although there is very low voltage in the limb leads (*ie*, amplitude of the QRS complex is < 5 mm in every limb lead). The axis is extremely negative between -30° and -90° (positive QRS complex in lead I and negative complex in leads II and aVF). Since the QRS complex has an rS morphology, this is termed a left anterior fascicular block. The QT/QTc intervals are slightly prolonged (420/470 msec). The T waves are inverted in leads I, aVL, and V2–V6. Although inverted, the T waves are symmetric (\neg). It is likely that the patient is still hyperkalemic, although the potassium level is certainly lower than when ECG 36A was obtained as the QRS complex duration is now normal. Although P waves are absent, suggesting that this is a junctional rhythm, it is possible that this is

a sinoventricular rhythm. This occurs as atrial activation has not yet been restored and P waves are not yet present.

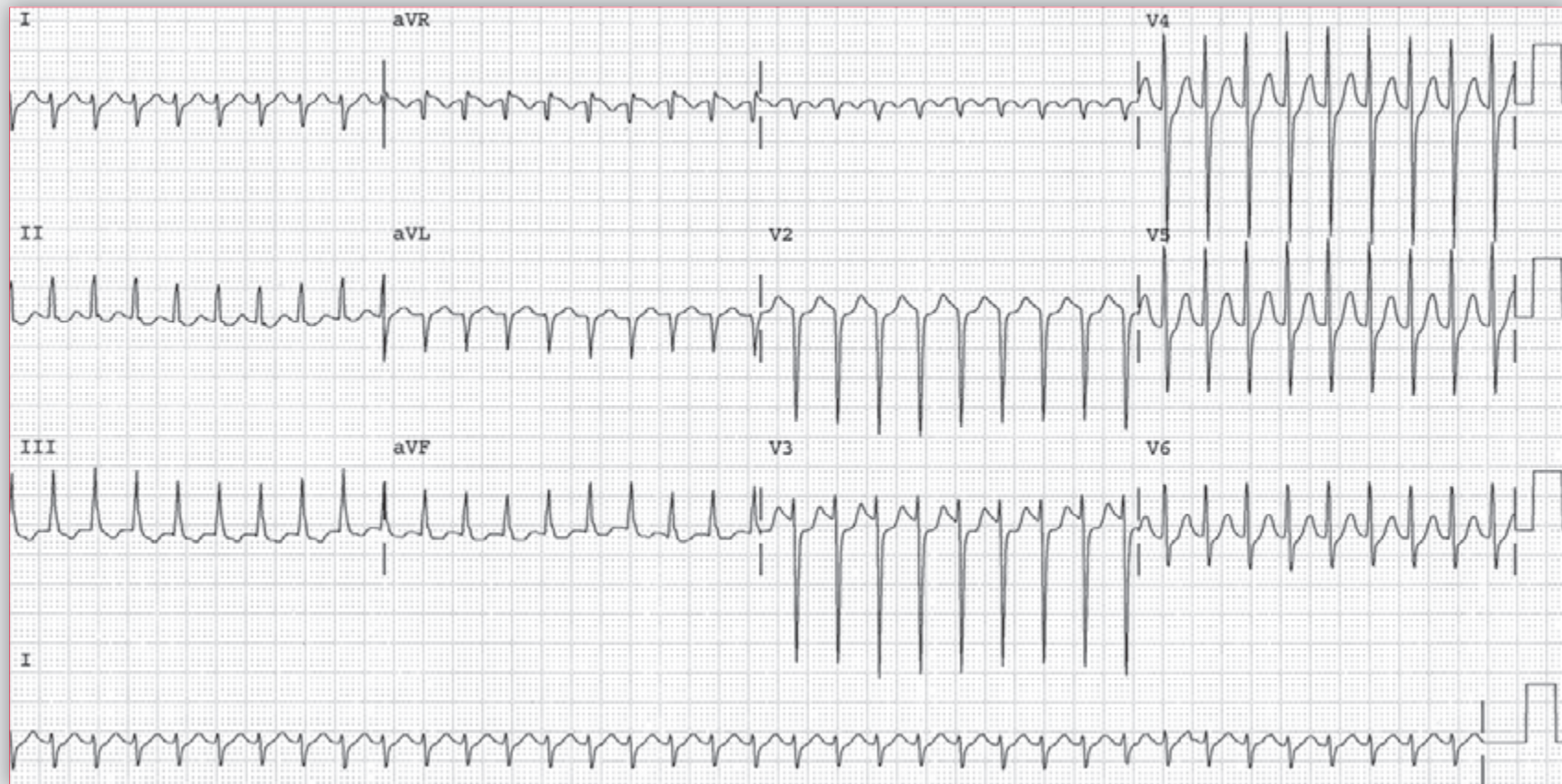
Acute therapy for hyperkalemia includes insulin (which forces potassium into the cells) and glucose (to prevent insulin-induced hypoglycemia). It has been recommended that calcium also be given to “stabilize the membrane” and enhance membrane excitability; however, how this happens is not understood, nor is it clear that this therapy is necessary. In addition a resin (Kayexelate) was given to remove potassium.

The clinical presentation in this patient is one of worsening heart failure, perhaps a result of an upper respiratory infection, with the development of acute on top of chronic renal insufficiency. As a result of the worsening renal failure, as well as therapy with an ACE inhibitor and spironolactone, acute hyperkalemia developed. ■

Core Case 37

A 22-year-old college student has the sudden onset of palpitations and lightheadedness while walking to his last class of the day. As a result he goes to the college health service clinic, where his pulse is noted to be over 200 bpm and blood pressure is 90/60 mm Hg.

ECG 37A



He states that he had a similar episode several months before, but that the symptoms resolved after a coughing spell. An ECG is obtained (ECG 37A).

After therapy, he immediately feels better, and an ECG is repeated (ECG 37B).

What does the first tracing (ECG 37A) show?

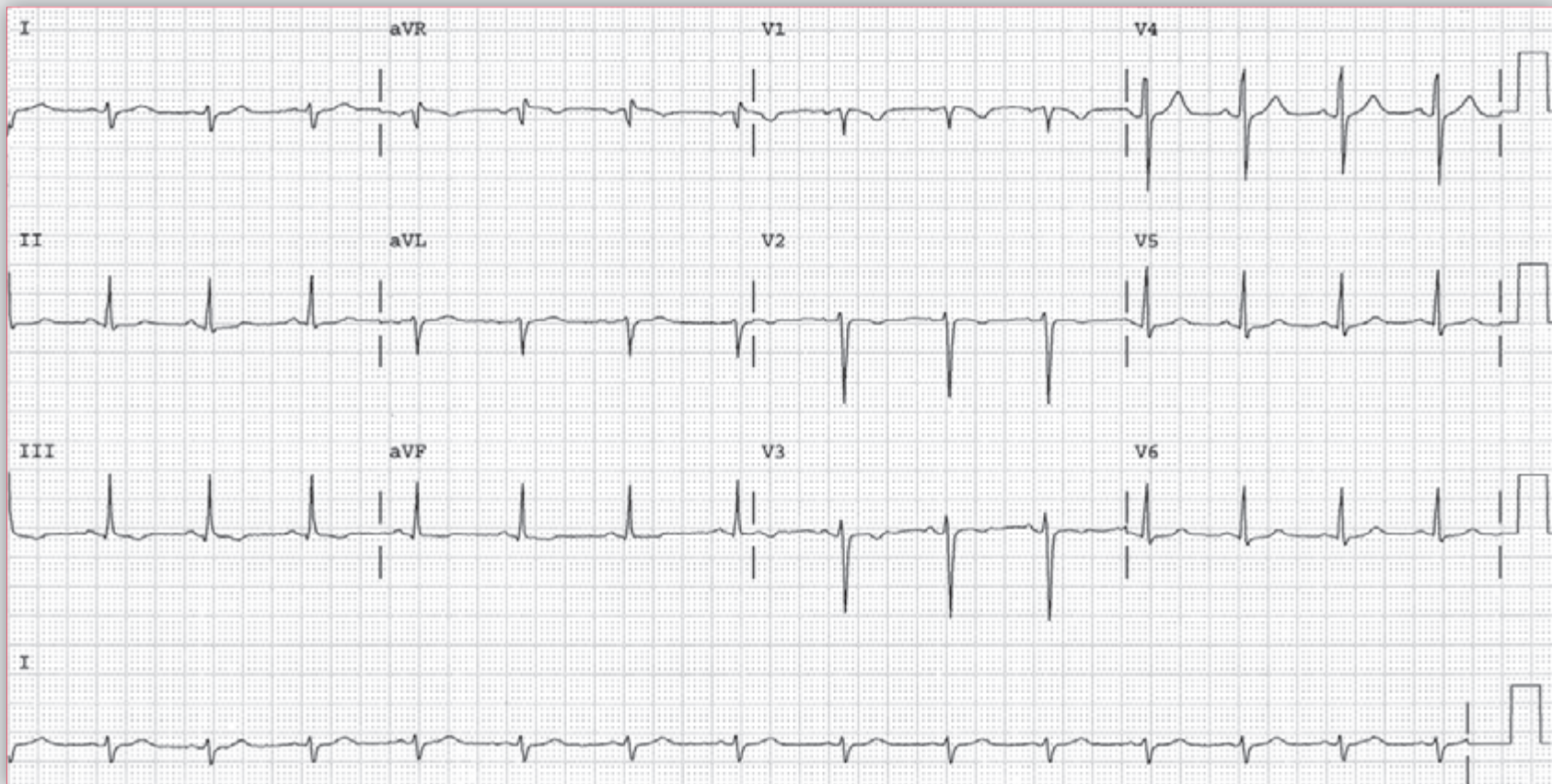
What is the most likely etiology?

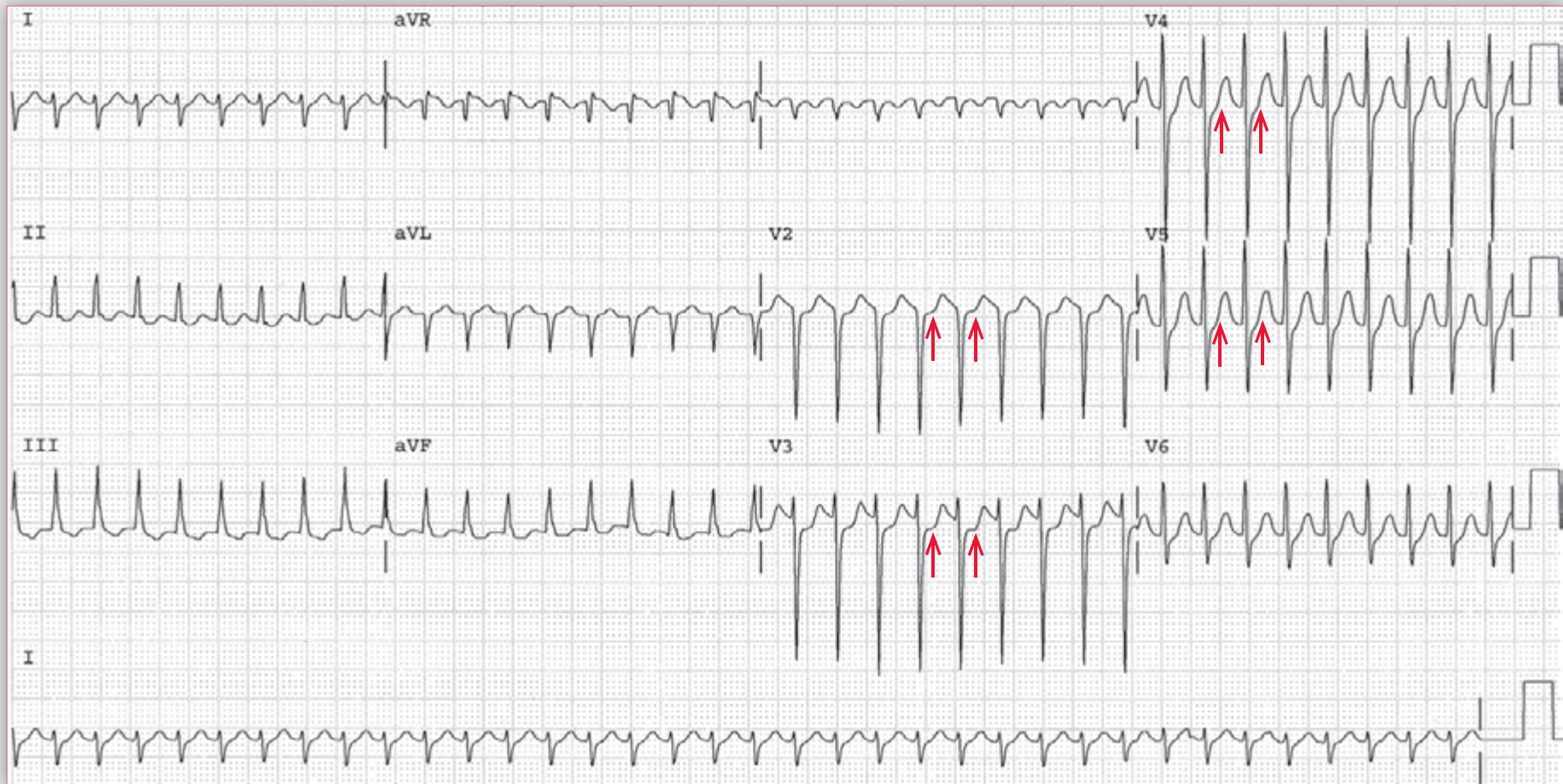
What therapy is indicated?

What does the second tracing (ECG 37B) show?

Is any further therapy needed?

ECG 37B





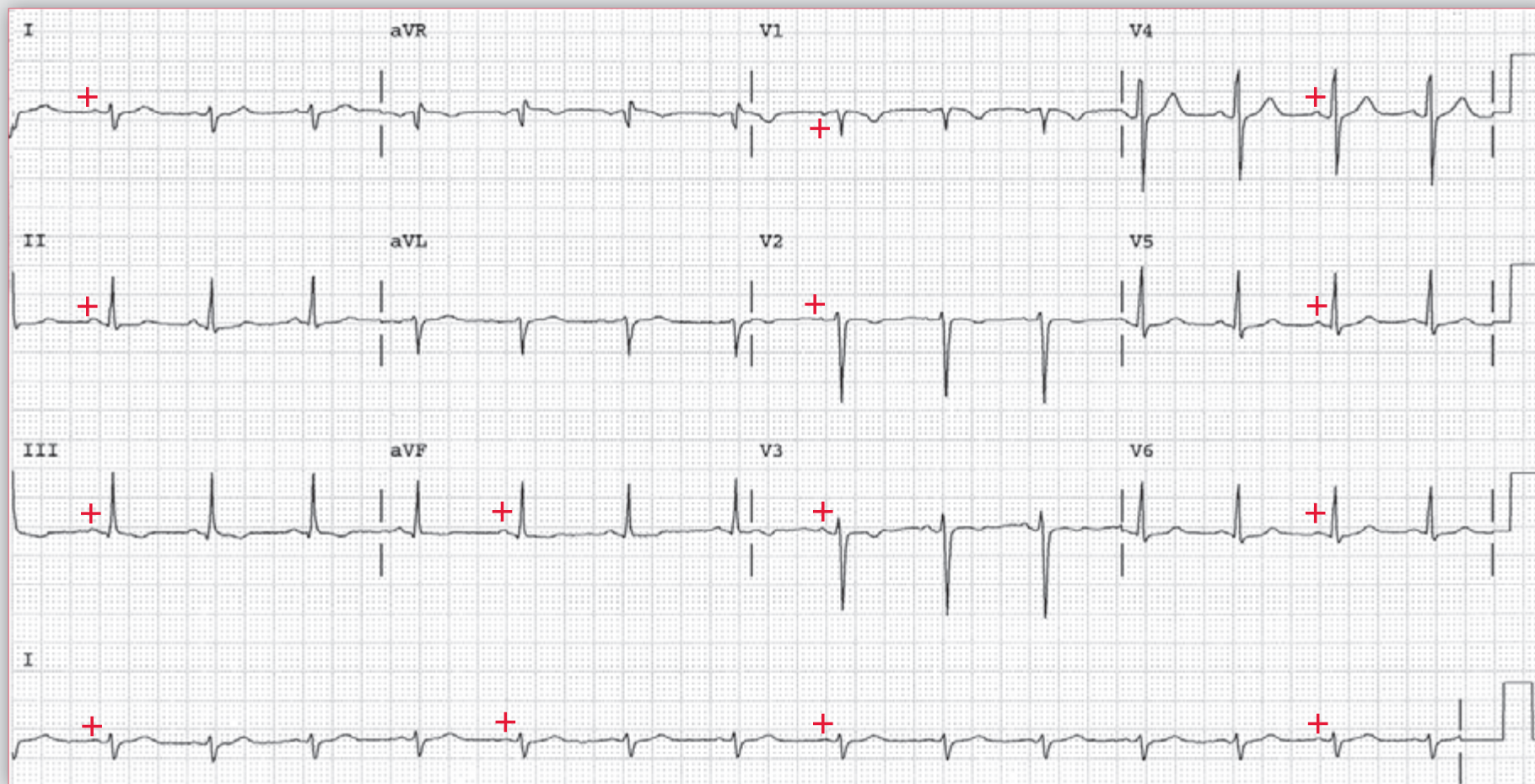
ECG 37A Analysis: Narrow complex supraventricular tachycardia, No-RP tachycardia, atrioventricular nodal reentrant tachycardia, left posterior fascicular block

ECG 37A shows a regular tachycardia at a rate of 220 bpm. There are no P waves seen before or after any of the QRS complexes. The QRS complexes have a normal duration (0.08 sec) and morphology. The axis is rightward between $+90^\circ$ and $+180^\circ$ (QRS complex positive in leads I and aVF). A rightward axis is seen with right ventricular hypertrophy (associated with a tall R wave in lead V1), a lateral wall myocardial infarction (associated with a Q wave in leads I and aVL), dextrocardia (negative P waves in leads I and aVL and reverse R wave progression across the precordium), Wolff-Parkinson-White (associated with a widened QRS complex due to a delta wave and a short PR interval) or R-L arm lead switch (associated with negative P wave in leads I and aVL). In the absence of any cause for the right axis, this is termed a left posterior fascicular block. Right ventricular hypertrophy and a lateral wall myocardial infarction are not present, and there is no

evidence for dextrocardia. Since P waves are not seen, lead switch cannot be established. Hence these are supraventricular QRS complexes. The QT/QTc intervals are normal (220/420 msec). There is upsloping ST-segment depression noted in leads V2–V5 (\uparrow). The ST segment is back to baseline at 0.08 second past the J point indicating that these are rate-related changes. The exception is lead V3 and the ST segment is still depressed at 0.08 second beyond the J point. As the ST-segment depression is localized to one precordial lead, it is not certain that this has any importance. There are also nonspecific ST-segment changes (flattening) in leads II, III, and aVF. In the absence of any obvious P waves, this supraventricular tachycardia is a no-RP tachycardia. The most common etiology for this is the common or typical type of an atrioventricular nodal reentrant tachycardia (slow-fast).

continues

Podrid's Real-World ECGs



ECG 37B Analysis: Normal sinus rhythm, left posterior fascicular block

ECG 37B is the baseline ECG for ECG 37A. There is a regular rhythm at a rate of 86 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.14 sec). The P wave is positive in leads I, II, aVF, and V4–V6; hence this is a normal sinus rhythm. The QRS complex duration, axis, morphology, and amplitude and QT/QTc intervals are identical to that in ECG 37A, confirming that the rhythm in ECG 37B is a no-RP tachycardia and hence an atrioventricular nodal reentrant tachycardia (AVNRT). The axis is rightward, similar to the axis in ECG 37A. As the P waves are seen and are positive in lead I, the right axis is not the result of R-L arm lead switch. Hence this is a left posterior fascicular block.

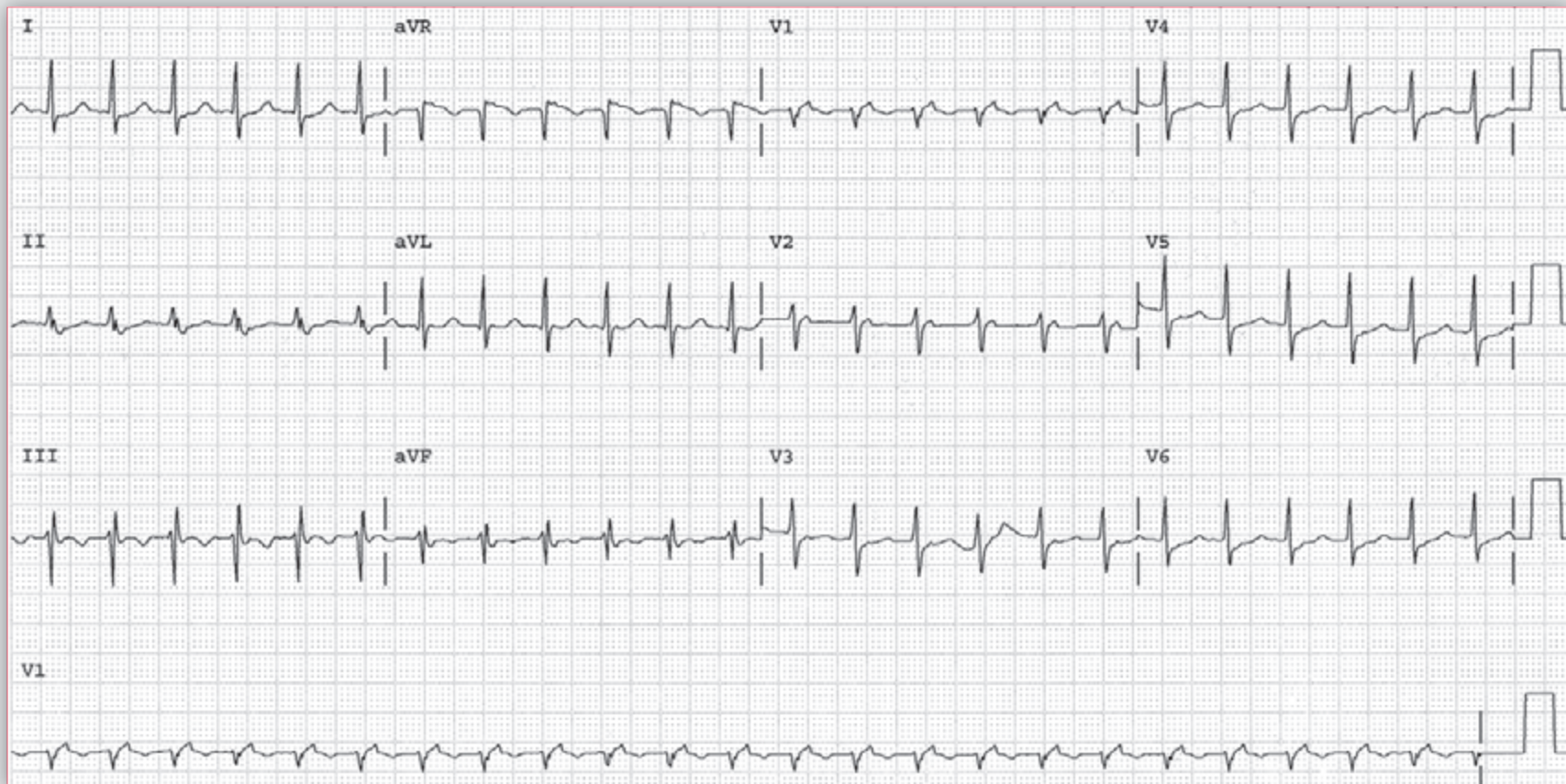
An AVNRT is due to a reentrant circuit within the AV node resulting from dual AV nodal pathways, *ie*, a fast pathway that conducts an impulse rapidly, but recovers (repolarizes) slowly, and a slow pathway

that conducts an impulse slowly, but recovers (repolarizes) rapidly. With typical AVNRT, the impulse conducts to the ventricle via the slow pathway while retrograde conduction to the atrium is via the fast pathway (hence termed slow-fast). As a result there is simultaneous atrial and ventricular activation and hence no P waves are seen. It can be terminated by any condition that alters the electrophysiologic properties of the AV nodal pathways. This can be achieved by vagal maneuvers, such as carotid sinus pressure, Valsalva, coughing, gagging, or vomiting. AV nodal agent is also effective and these include adenosine, a β -blocker, a calcium-channel blocker (verapamil or diltiazem), or digoxin. The need for long-term therapy to prevent recurrence depends upon the frequency of episodes, associated symptoms and if the arrhythmia can be easily terminated, such as with a vagal maneuver. Therapy, if needed, consists of a β -blocker, calcium-channel blocker, or digoxin. AV nodal ablation using radiofrequency waveforms is also an effective approach. ■

Core Case 38

A 19-year-old woman has a history of intermittent palpitations presents to the emergency department after developing the sensation of a racing heart before a college exam.

ECG 38A

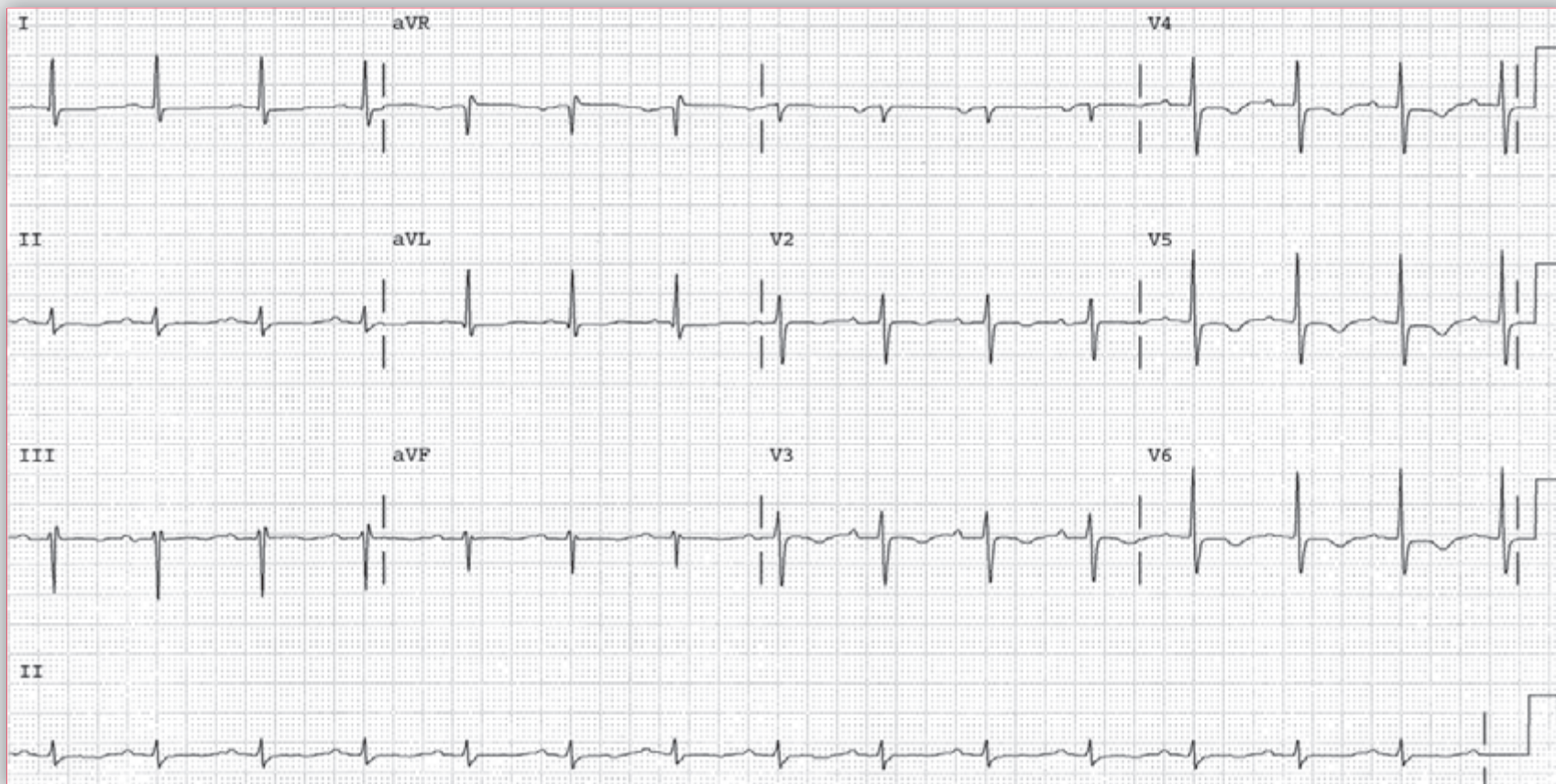


An initial ECG (ECG 38A is obtained).
While waiting to be seen her symptoms resolve
spontaneously and a follow-up ECG is obtained
(ECG 38B).

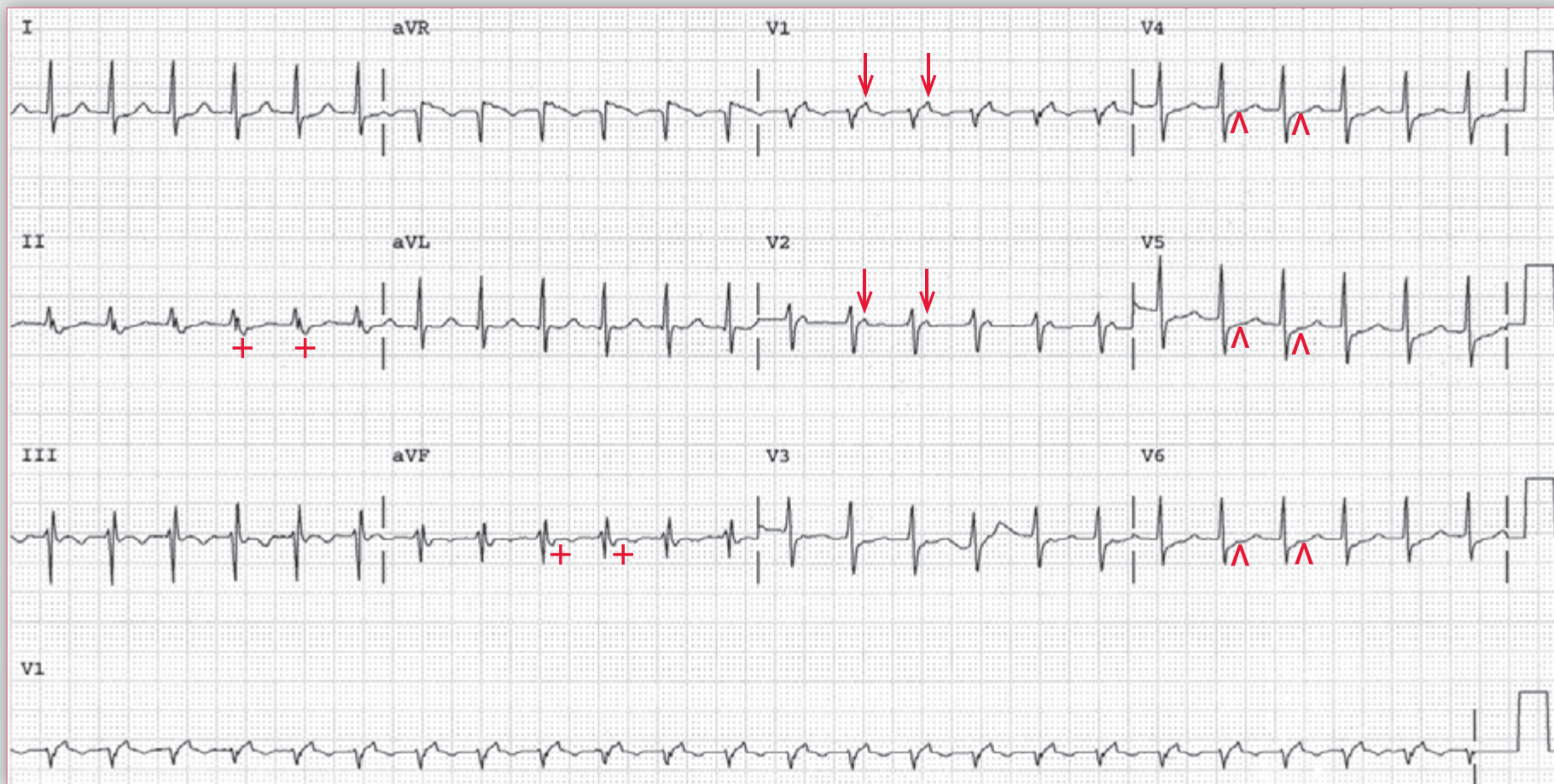
What does the ECG show?

What is the differential diagnosis?

ECG 38B



Podrid's Real-World ECGs



ECG 38A Analysis: Narrow complex supraventricular tachycardia (short-RP type)

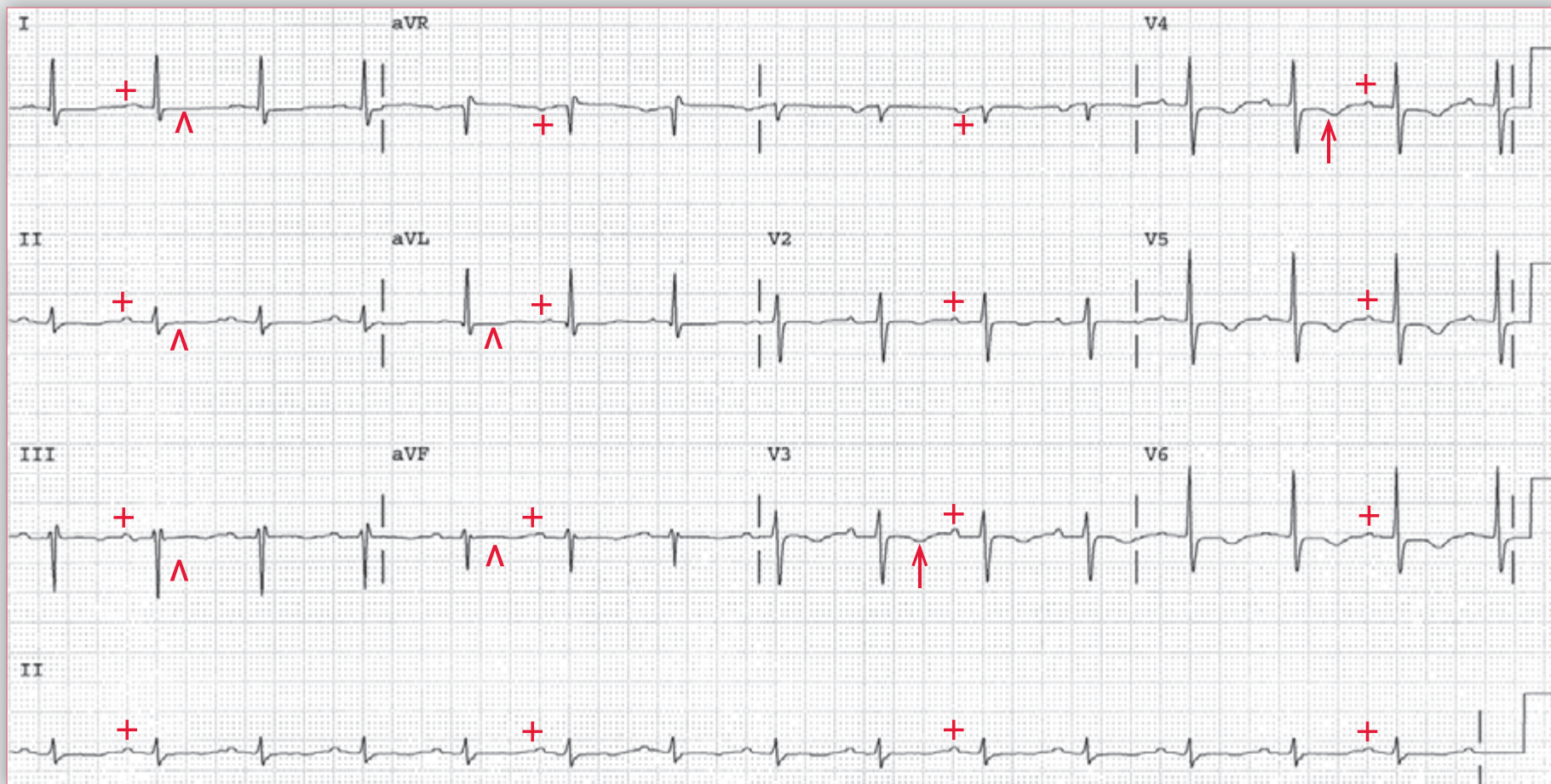
ECG 38A shows a regular rhythm at a rate of 140 bpm. The QRS complex duration is normal (0.08 sec) and there is a physiologic leftward axis between 0° and -30° (positive QRS complex in leads I and II and negative in aVF). The QRS morphology is normal. The QT/QTc intervals are normal (280/430 msec). There are no P waves before any of the QRS complexes. There is a positive waveform after the QRS complex, best seen in leads V1–V2 (\downarrow). This appears to be a P wave. It is distinct from the QRS complex and within the initial portion of the ST segment (*ie*, RP interval is > 0.08 second but less than half the RR interval. This P wave can also be seen in leads V4–V6 as a notching of the ST segment (\wedge) and also in leads II and aVF where it appears to be an S wave ($+$). The RP interval (\sqcap) is 0.10 second and the PR interval is 0.30 second. Hence this is a short-RP tachycardia.

There are a number of etiologies for a short-RP tachycardia, including an atrioventricular reentrant tachycardia (AVRT), atrial tachycardia, a variant of a common atrioventricular nodal reentrant tachycardia (AVNRT) termed slow-slow, a sinus tachycardia with a first-degree AV

block, atrial flutter with 2:1 AV conduction, or an ectopic junctional tachycardia. As the P wave appears to be negative in leads II and aVF, this is not likely to be sinus rhythm. There is no evidence for a second atrial waveform and hence this is not likely to be atrial flutter. The differential includes atrial tachycardia or a rhythm from the AV node or junction (*ie*, junctional tachycardia, AVRT, or AVNRT). Unfortunately, the ECG does not provide any additional information to establish the etiology. However, at this rate an ectopic junctional tachycardia is uncommon. An unusual form of an AVNRT (slow-slow) is also unusual in young people, as it usually reflects relatively slow conduction back to the atrium via the fast pathway; this is more commonly seen in older subjects or in those on AV nodal drugs. Hence the two most likely etiologies would be an atrial tachycardia or an AVRT (either due to an overt bypass tract, *ie*, bundle of Kent with Wolff-Parkinson-White or bundle of James in Lown-Ganong-Levine, or a concealed bypass tract).

continues

Podrid's Real-World ECGs



ECG 38B Analysis: Normal sinus rhythm with first-degree AV block, nonspecific ST-T wave changes

ECG 38B is the baseline ECG for ECG 38A. There is a regular rhythm at a rate of 86 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.22 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus rhythm with a first-degree AV block (first degree AV conduction delay). The QRS complex duration, axis, and morphology are identical to that seen in ECG 38A. Noted is that the positive waveform after the QRS complex in leads V1–V2 is not present, there is no S wave in leads II and aVF, and there is no notching

of the ST segment in leads V4–V6. This supports the fact that these abnormalities seen in ECG 38A were P waves and hence it is a short-RP tachycardia. There is no evidence for a preexcitation syndrome (*ie*, PR interval is long), and therefore, the arrhythmia in ECG 38A is either an atrial tachycardia or an AVRT due to a concealed bypass tract.

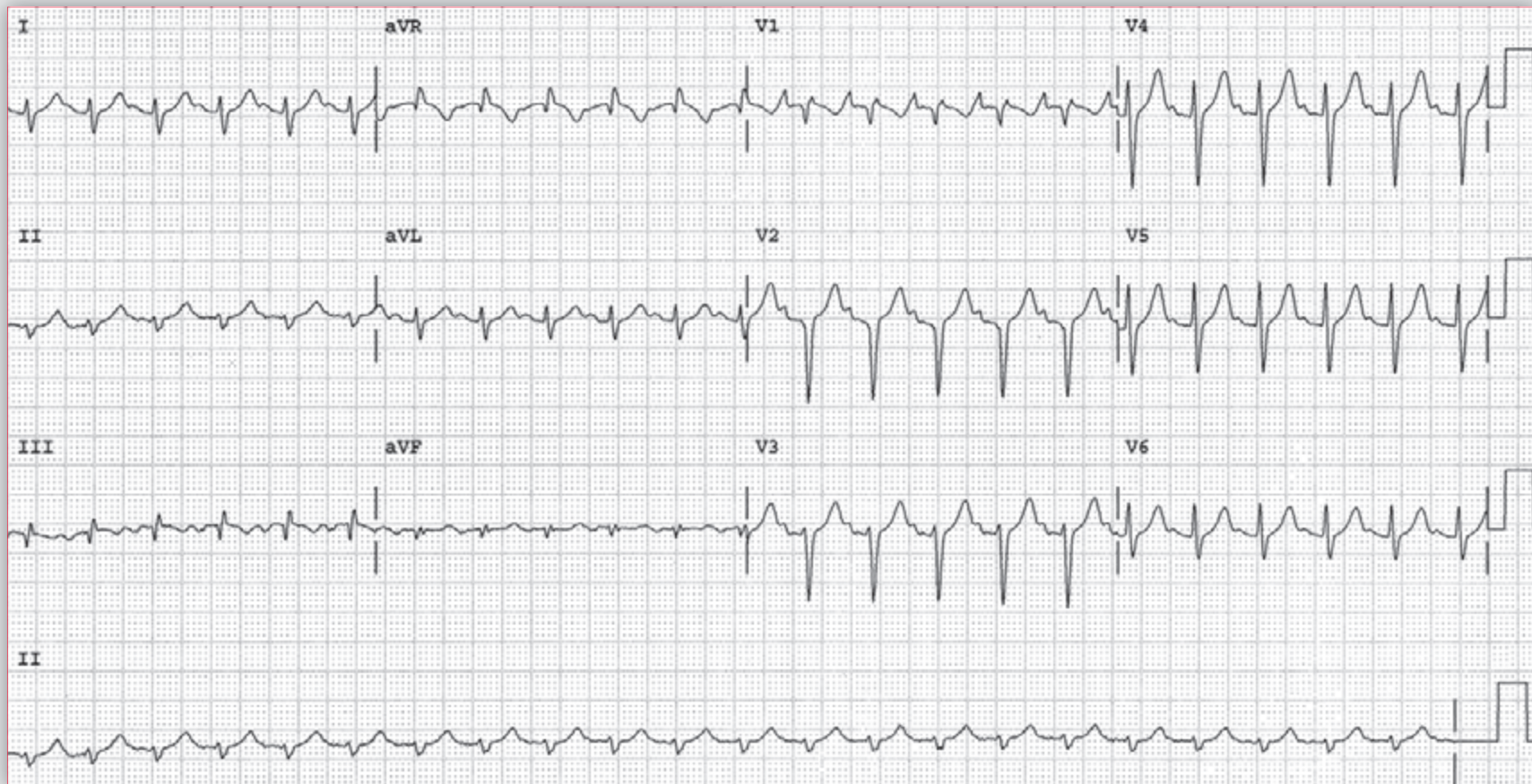
Also noted are diffuse T-wave abnormalities, *ie*, flattening in the limb leads (^) and inversion (↑) in leads V3–V6. ■

Notes

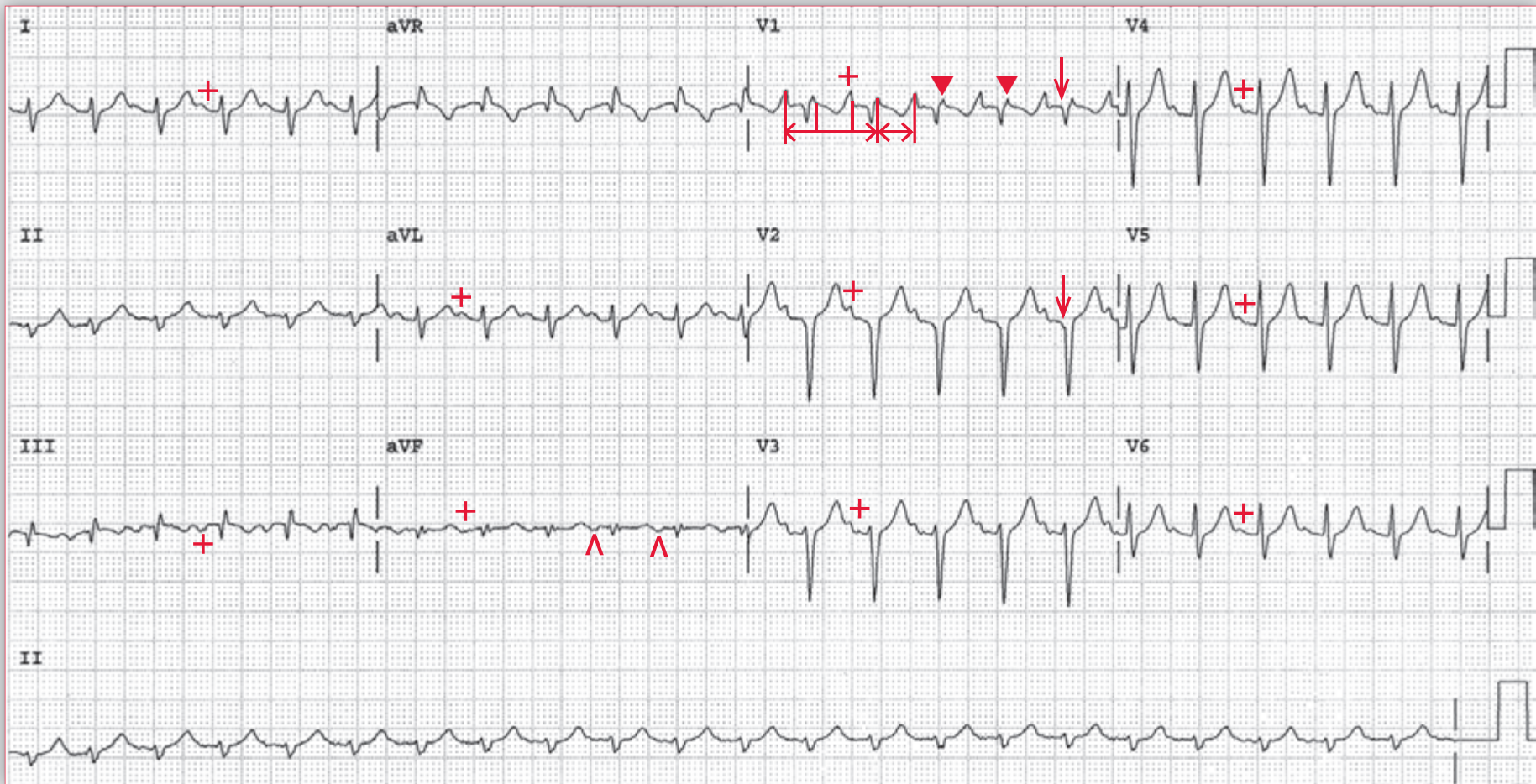
A 64-year-old man has had intermittent palpitations over the past few years. They typically last 5 to 10 minutes and resolve spontaneously. He presents to the emergency department when an episode last for more than an hour.

What does the ECG show?

What is the differential diagnosis?



Podrid's Real-World ECGs



ECG 39 Analysis: Narrow complex supraventricular tachycardia (long-RP type), poor R-wave progression in leads V1–V2, consistent with a previous anteroseptal myocardial infarction, left posterior fascicular block, low voltage in limb leads

ECG 39 shows a regular rhythm at a rate of 140 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.16 sec). The P wave is positive in leads I, II, V4–V6; however, it is negative in lead aVF. There is another waveform seen at the end of the QRS complex in lead V1 (▼). Although it is similar to the P wave, the intervals between the P wave and this waveform (0.20 sec) (⊐) are not the same as the interval between this waveform and the following P wave (0.26 sec) (↔). Therefore, this is not another on-time atrial waveform, but is part of the QRS complex, *ie*, an R' representing a right ventricular conduction delay. Hence this is a long-RP tachycardia (RP = 0.28 sec, PR = 0.16 sec). Etiologies for a long-RP tachycardia include sinus tachycardia, atrial tachycardia, ectopic junctional tachycardia, atrial flutter with 2:1 AV block, atrioventricular reentrant tachycardia (AVRT), and atypical (fast-slow) atrioventricular nodal reentrant tachycardia (AVNRT) (*ie*, conduction to the ventricles is via the fast pathway while retrograde atrial activation is via the slow pathway). The P waves are negative in lead aVF (^); hence this is not a sinus tachycardia. There is no evidence for a second on-time atrial waveform; hence this is not atrial flutter. There are no other features on the ECG that would be useful to distinguish atrial tachycardia from a junctional

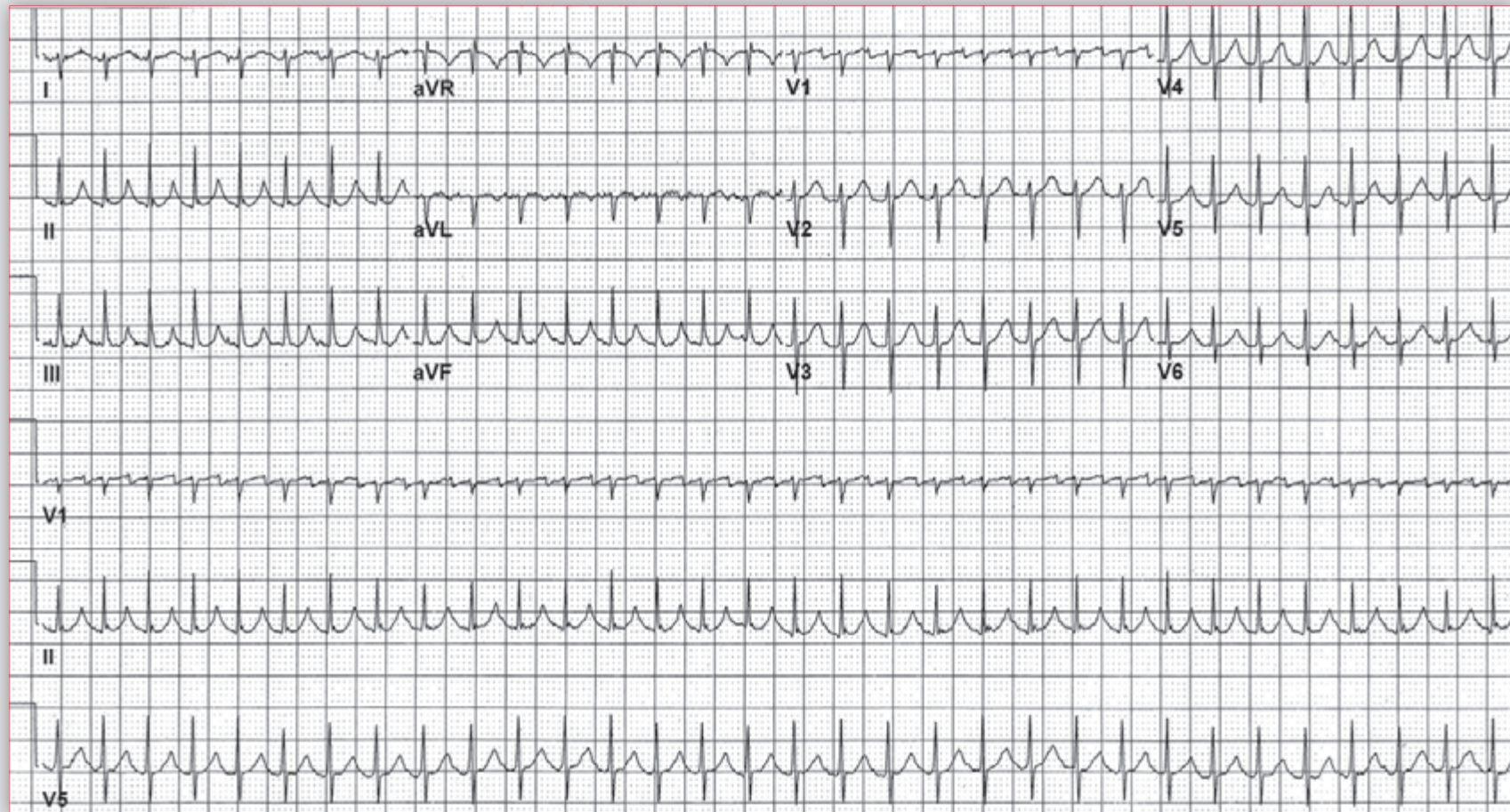
tachycardia (*ie*, ectopic, AVNRT, or AVRT) as each of these arrhythmias is associated with a negative P wave in lead aVF. However, the most common etiology would be an atrial tachycardia.

The QRS complex duration is normal (0.08 sec) and there is a rightward axis between +90° and +180° (QRS complex negative in lead I and positive in lead aVF). There are a number of etiologies for a rightward axis including right ventricular hypertrophy (with tall R wave in lead V1 and P pulmonale), an old lateral wall myocardial infarction (with Q waves in leads I and aVF), Wolff-Parkinson-White (with short PR interval and delta wave), R-L arm lead switch (with negative P waves and T waves in leads I and aVL), dextrocardia (resembles R-L arm lead switch and reverse R-wave progression across the precordium) or a left posterior fascicular (with an rS morphology in lead I and aVL), which is a diagnosis of exclusion. In this case this is a left posterior fascicular block. The voltage in the limb leads is low (< 5 mm in each lead). There are Q waves (QS complexes) in leads V1–V2 (↓), consistent with a previous anteroseptal myocardial infarction. The QT/QTc intervals are normal (280/430 msec). ■

Core Case 40

A 38-year-old man is admitted to the hospital with acute pancreatitis. In the first 6 hours of his hospitalization, he is noted to be very tachycardic with heart rates of over 200 bpm. Blood pressure is 80/60. An ECG is obtained (ECG 40A), and another is obtained after resuscitation with intravenous fluids and pain control (ECG 40B).

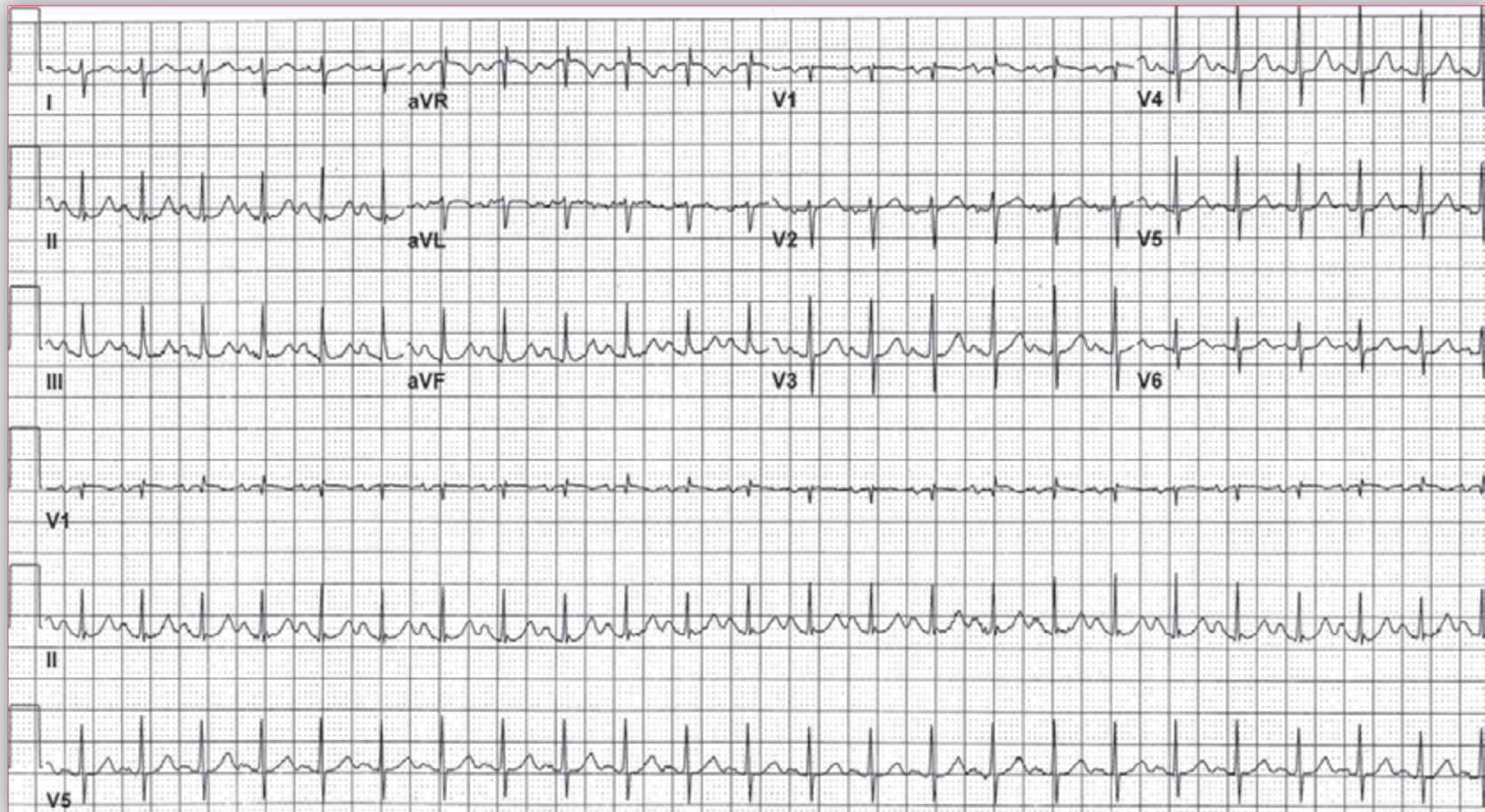
ECG 40A



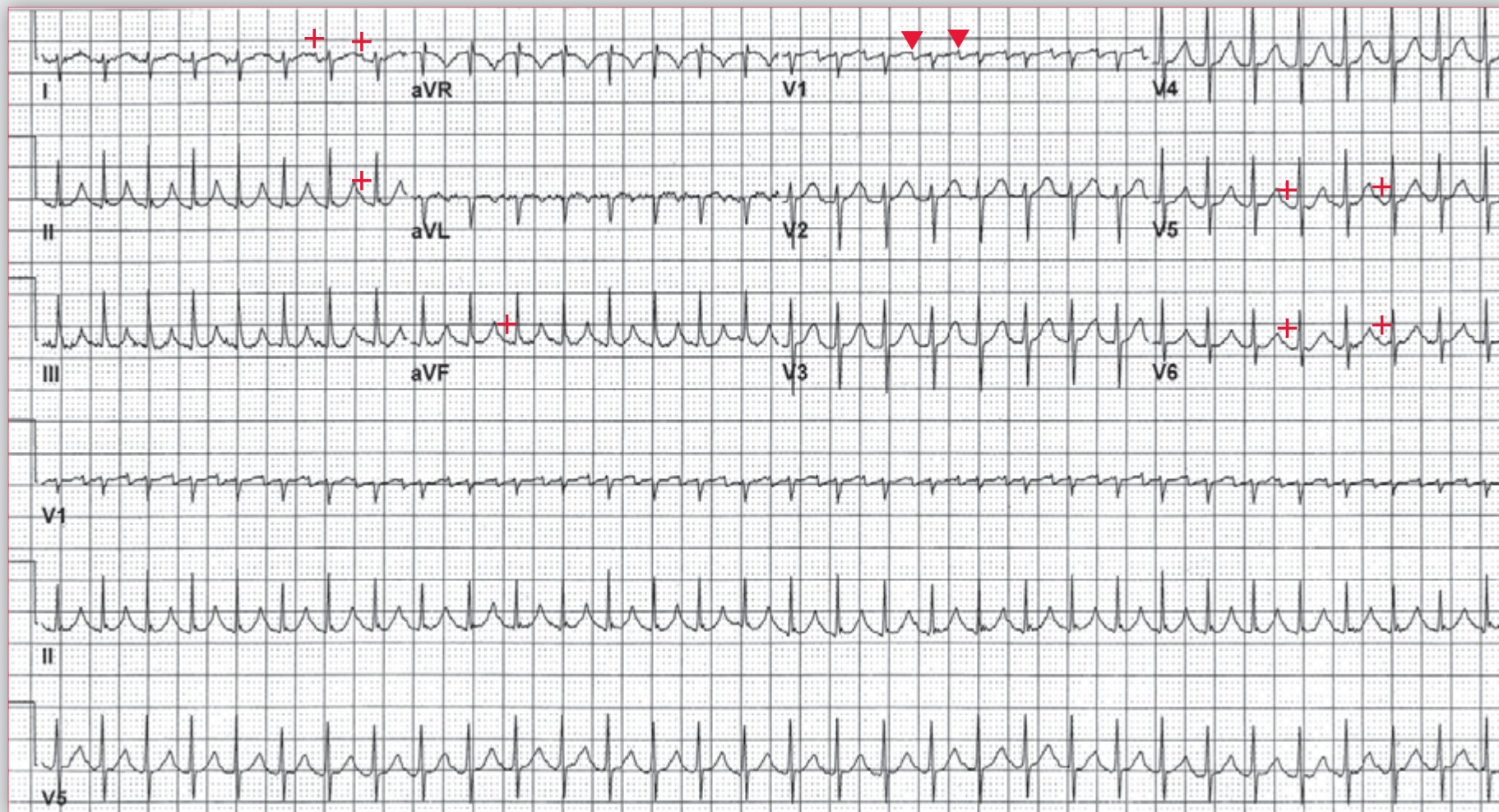
What do the ECGs show?

What is the likely diagnosis?

ECG 40B



Podrid's Real-World ECGs

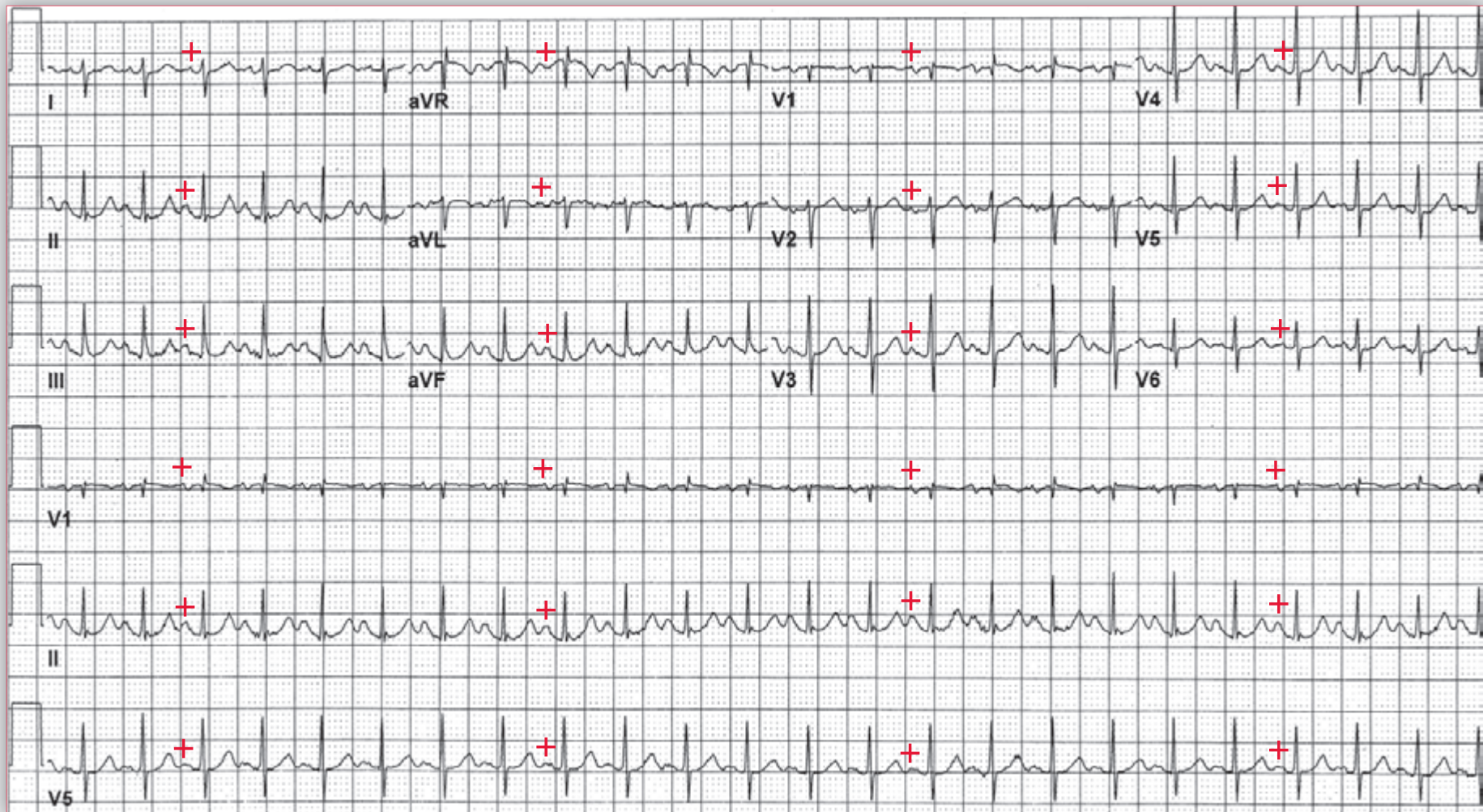


ECG 40A Analysis: Narrow complex supraventricular tachycardia
(long-RP tachycardia) (sinus tachycardia), left posterior fascicular block

ECG 40A shows a regular rhythm at a rate of 210 bpm. The QRS complex duration is normal (0.08 sec) and there is a normal morphology. The axis is rightward between $+90^\circ$ and $+180^\circ$ (QRS complex negative in lead I and positive in aVF). Etiologies for a rightward axis include a lateral wall myocardial infarction (Q waves in leads I and aVL), right ventricular hypertrophy (tall R wave in lead V1), dextrocardia (negative P and T waves in leads I and aVL and reverse R wave progression across the precordium), R-L arm lead switch (negative P and T waves in leads I and aVL), Wolff-Parkinson-White (short PR interval and delta wave), or a left posterior fascicular block (which is a diagnosis of exclusion). The QT/QTc intervals are normal (240/450 msec). Although P waves are not obvious, careful inspection of leads I, II, aVF, and V5–V6 reveals small positive deflections at the end of the T wave (+).

These are probably P waves. The PR interval is 0.12 second. There is a prominent waveform seen in lead V1 (▼), which has the same PR interval as that seen in leads I and V5–V6. This waveform is a P wave and not the T wave. Hence this is a long-RP tachycardia with a PR interval of 0.12 second and an RP interval of 0.20 second. Etiologies include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atypical or uncommon atrioventricular nodal reentrant tachycardia (fast-slow), or atrioventricular reentrant tachycardia. The P wave appears to be positive in leads I, II, aVF, and V5–V6 and biphasic in lead V1, suggesting that this likely a sinus tachycardia. As there is no evidence for a lateral wall MI, right ventricular hypertrophy, dextrocardia, Wolff-Parkinson-White, or R-L arm lead switch, the right axis is due to a left posterior fascicular block.

continues



ECG 40B Analysis: Sinus tachycardia, left posterior fascicular block

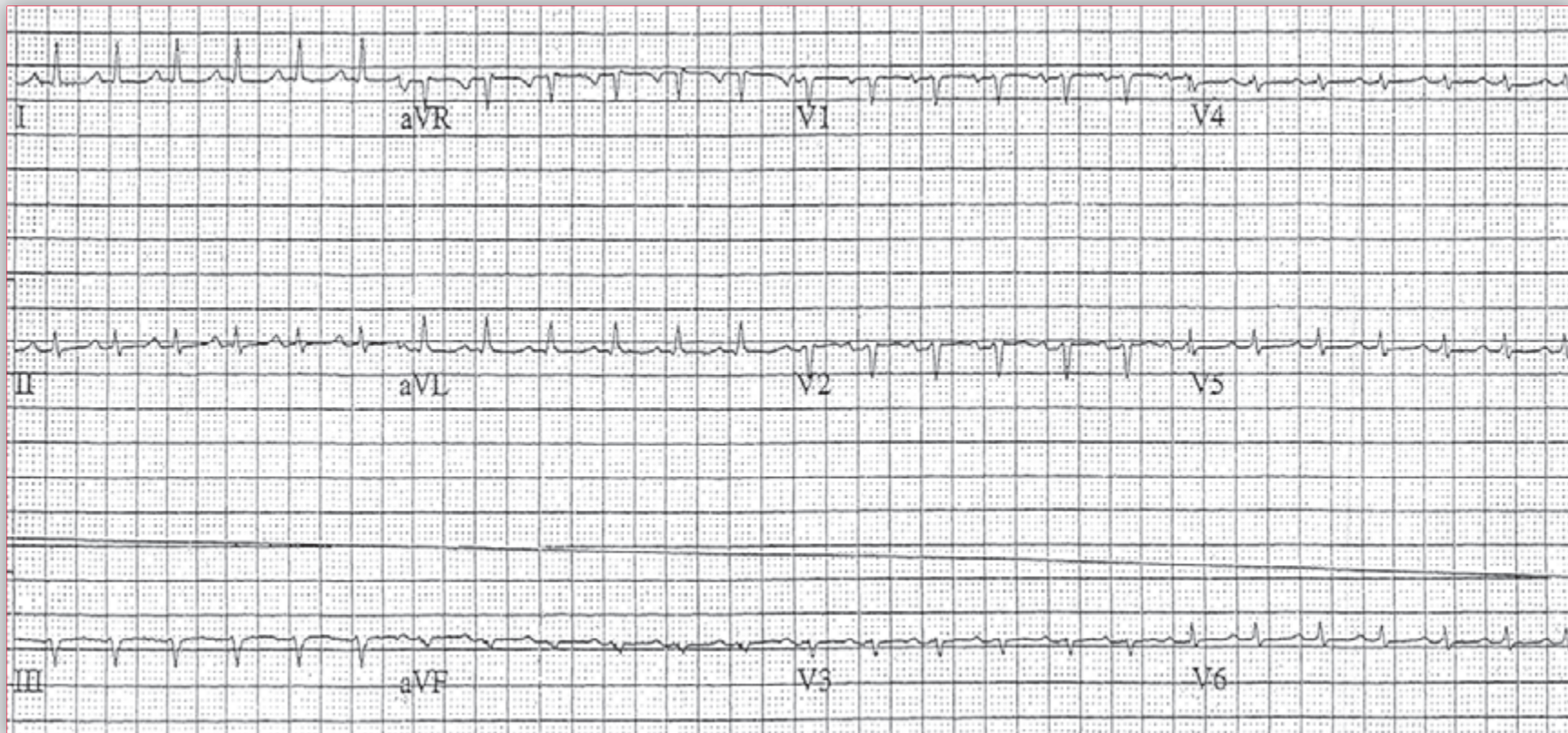
ECG 40B is from the same patient as ECG 40A. There is a regular rhythm at a rate of 144 bpm. The QRS duration, axis, and morphology are identical to that seen on ECG 40A. There is a distinct P wave (+) seen before each QRS complex with a stable PR interval (0.14 sec). The P wave is positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. The P-wave morphology is the same as was noted in ECG 40A, confirming that the etiology of the long-RP tachycardia on ECG 40A is sinus tachycardia. The slightly longer PR interval is related to the slower sinus rate.

Physiologic sinus tachycardia is always the result of sympathetic stimulation. As a result, there is a shortening of the PR interval as sympathetic stimulation enhances conduction through the AV node. This accounts for the short PR interval at a rate of 220 bpm and a slightly longer PR interval when the rate is slower at 144 bpm. In this case, the sinus tachycardia is the result of dehydration with hypotension and pain. ■

Core Case 41

A 64-year-old woman is admitted to the hospital for elective orthopedic surgery. Before her operation, she is noted to have periods of tachycardia that are sudden in their onset and offset. She feels well and is unaware of these episodes. An ECG is obtained (ECG 41A) as well as a rhythm strip during termination of one of these episodes (ECG 41B).

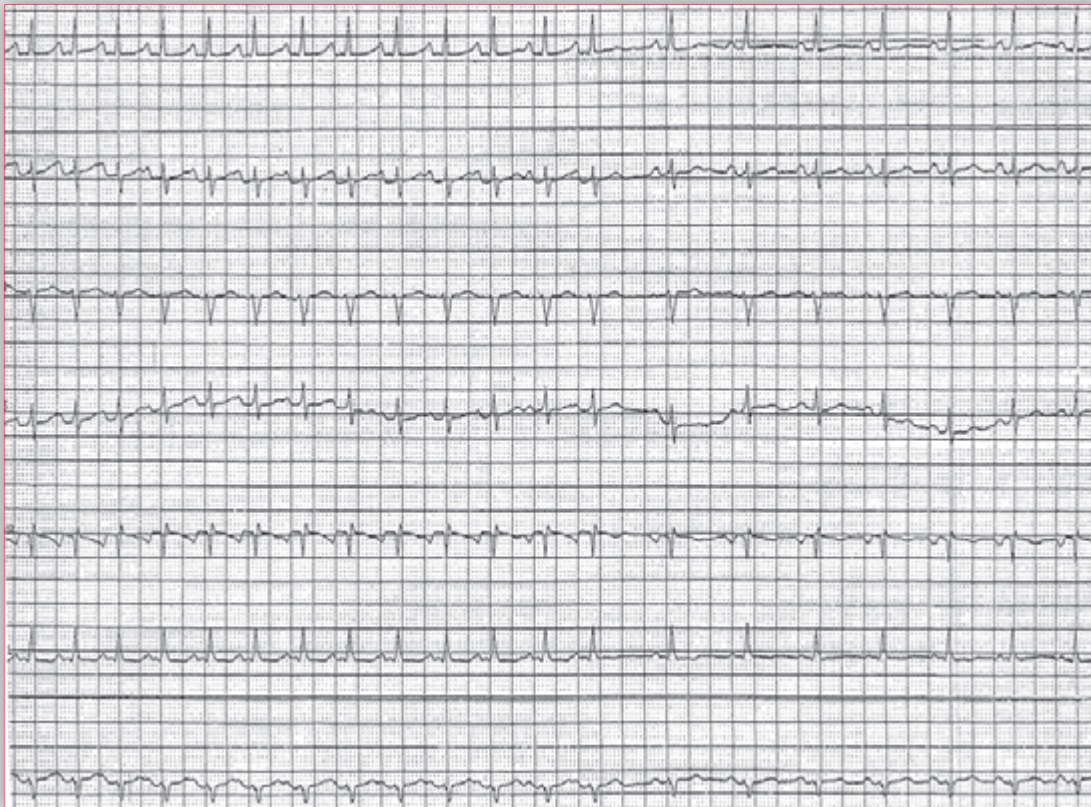
ECG 41A



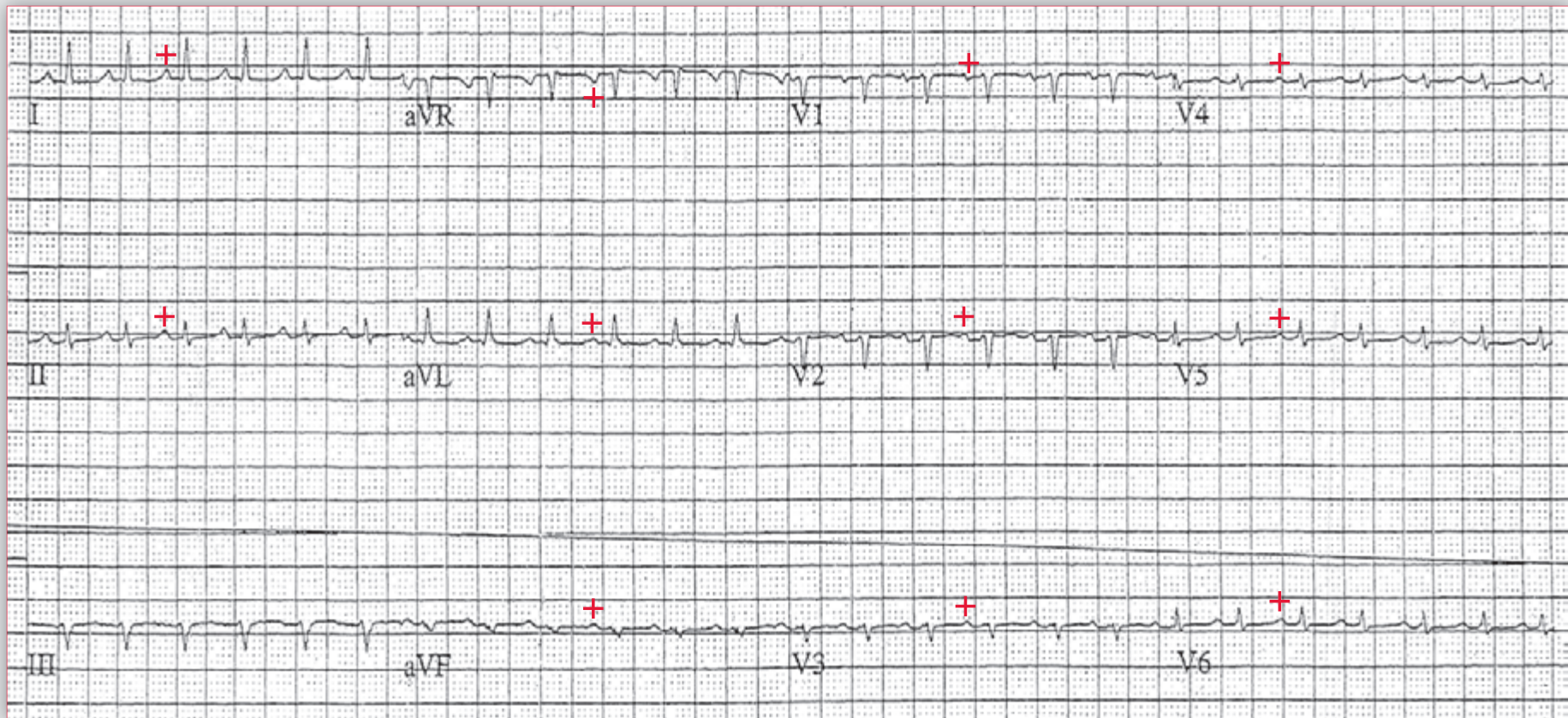
What do the tracings show (ECG 41A and ECG 41B)?

What is the likely diagnosis?

ECG 41B



Podrid's Real-World ECGs

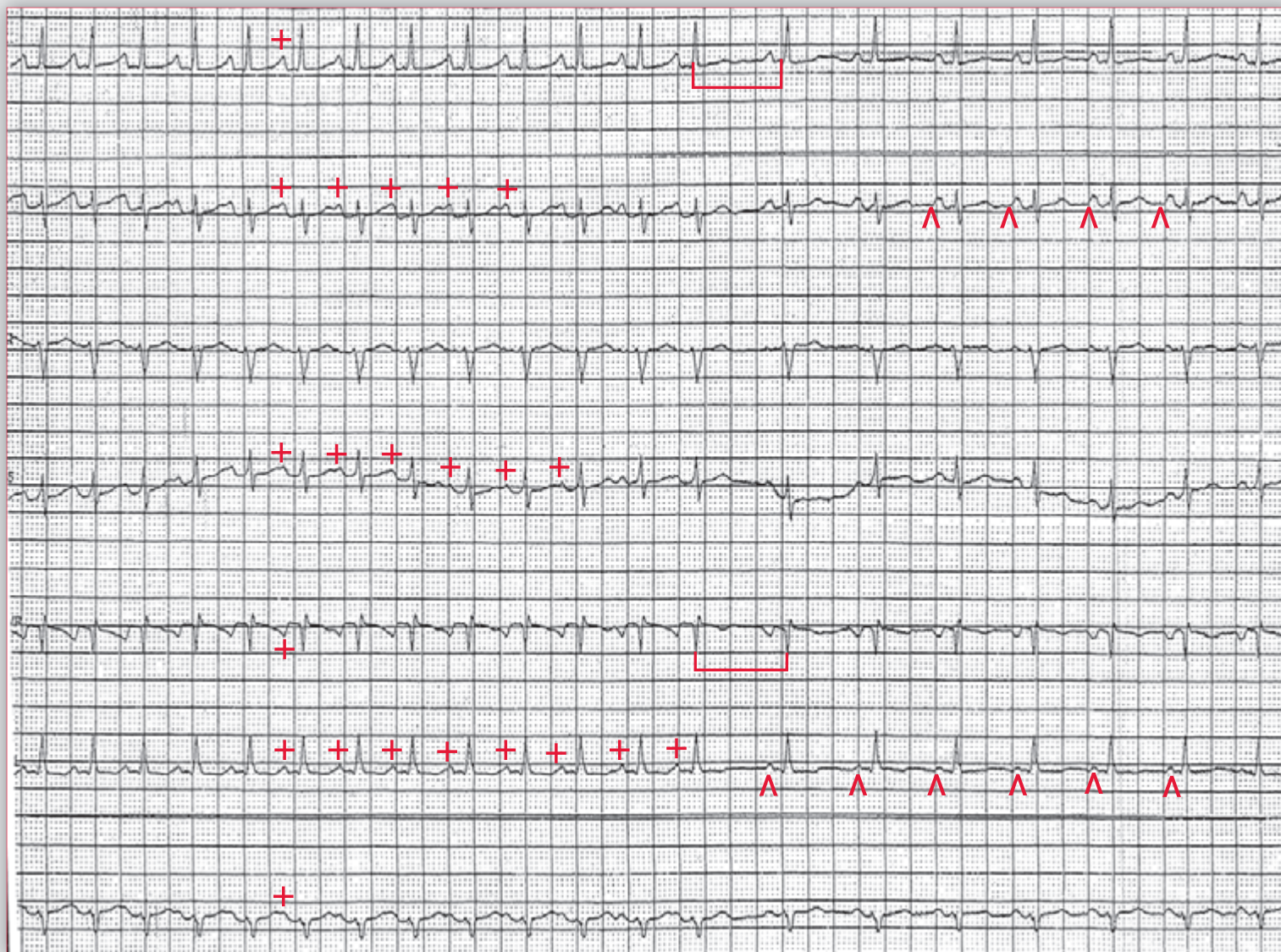


ECG 41A Analysis: Sinus tachycardia, low QRS voltage, left axis

ECG 41A shows a regular rhythm at a rate of 148 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. The QRS complex duration is normal (0.08 sec) and

there is a leftward axis between 0° and -30° (positive QRS complex in lead I and negative in leads II and aVF). There is low QRS complex voltage (< 5 mm in each limb lead and < 10 mm in each precordial lead). The QT/QTc intervals are normal (260/410 msec). *continues*

Podrid's Real-World ECGs



ECG 41B Analysis: Sinus node reentry

ECG 41B is a series of rhythm strips from the same patient as ECG 41A. The first 13 QRS complexes are regular at a rate of 148 bpm, which is the same as seen in ECG 41A. The QRS complex morphology and axis are identical to those seen in ECG 41A. The QT/QTc intervals are normal and the same as in ECG 41A. There is a P wave (+) before each of the QRS complexes with a stable PR interval (0.16 sec). The P wave and PR interval are identical to those seen in ECG 41A. Therefore, this is a sinus tachycardia. After the thirteenth QRS complex, there is an abrupt slowing of the rate (⏏) to 96 bpm. There is a P wave (^) before each of the QRS complexes and the PR interval is stable (0.16 sec).

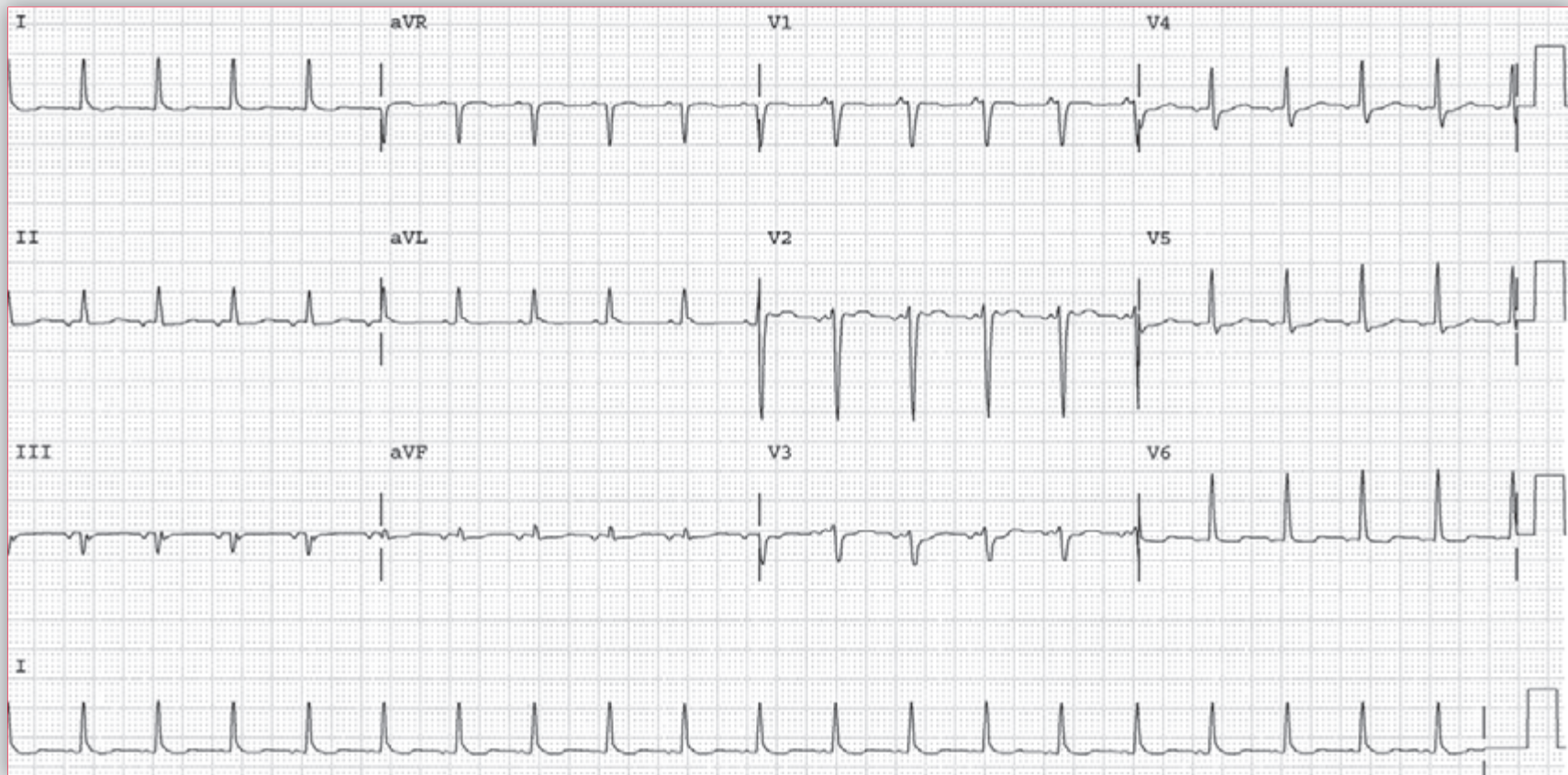
The P-wave morphology and PR interval are identical to those in complexes 1 through 13. Hence there is an abrupt termination of sinus tachycardia to a normal sinus rhythm. This is consistent with sinus node reentry. This arrhythmia resembles sinus tachycardia. However, in sinus tachycardia the heart rate gradually increases and then gradually decreases. In this case, there was an abrupt termination of the sinus tachycardia. This is the way reentrant arrhythmias terminate. Hence this is a sinus node reentry, which results from a circuit that involves the sinus node as well as the perinodal tissue. ■

Notes

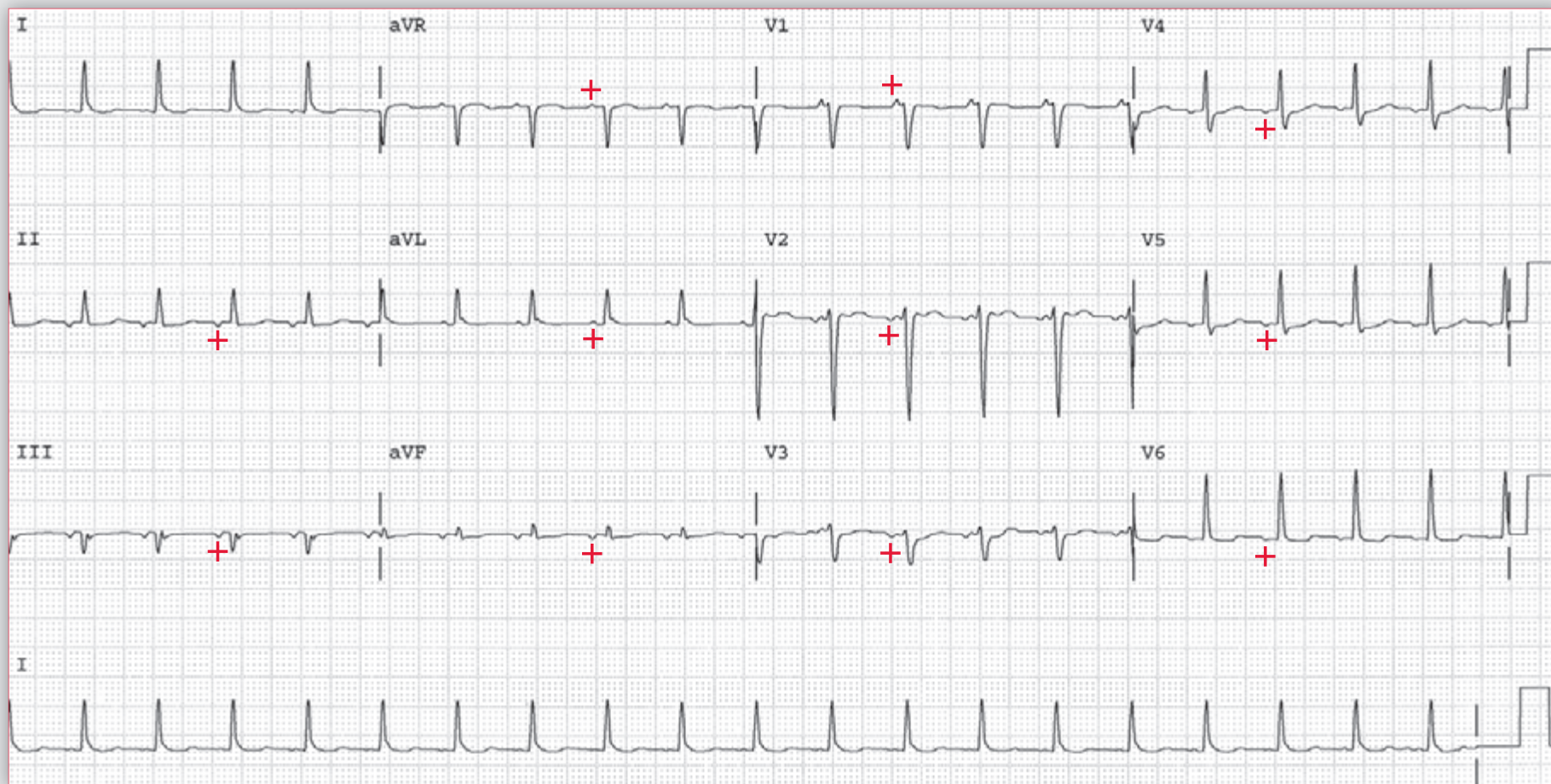
An 88-year-old man is hospitalized with pneumonia. He is noted to have an elevated heart rate that persists throughout his treatment. He feels well. His oxygenation is normal, he is afebrile, and in no respiratory distress.

What does the ECG show?

What is the likely diagnosis?



Podrid's Real-World ECGs



ECG 42 Analysis: Narrow complex supraventricular tachycardia (long-RP tachycardia), atrial tachycardia, clockwise rotation

ECG 42 shows a regular rhythm at a rate of 120 bpm. There is a P wave (+) before each QRS complex with a constant, but short, PR interval (0.10 sec). The P wave is negative in leads II, aVF, and V4–V6; the P wave is not of sinus origin. Hence this is a long-RP tachycardia (PR interval = 0.36 sec). Etiologies for a long-RP tachycardia include sinus tachycardia, atrial tachycardia, ectopic junctional tachycardia, atrial flutter with 2:1 AV block, atrioventricular reentrant tachycardia, or atypical atrioventricular nodal reentrant tachycardia (fast-slow). This arrhythmia has characteristics most suggestive of an ectopic atrial tachycardia given the rate and the length of the RP interval. The QRS complex duration is normal (0.08) and the R-wave progression in leads V1–V3 is slow, suggesting clockwise rotation of the electrical axis in the horizontal plane. This is determined by imagining the heart as viewed from under the diaphragm. With clockwise rotation, the left

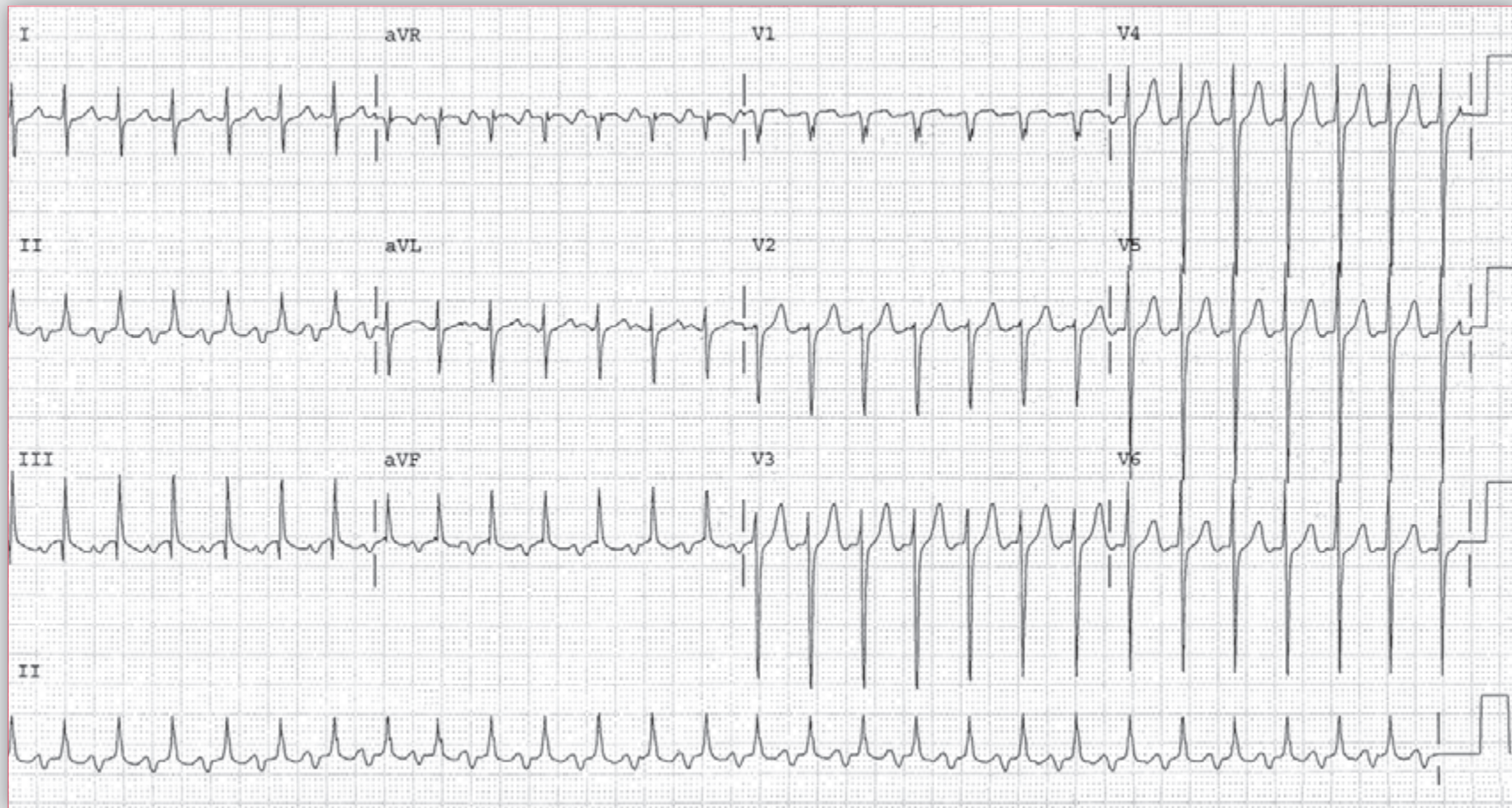
ventricular forces develop late in the precordial leads. The QT/QTc intervals are normal (280/400 msec). The T waves show low amplitude in most leads; this is a nonspecific abnormality.

An atrial tachycardia is most often the result of an ectopic atrial focus that may become activated under a number of circumstances. A frequent cause is pulmonary, and in this case, a pneumonia. Infrequently the mechanism is reentry, with a small circuit within the atrial myocardium. It is impossible to establish the actual mechanism on the surface ECG, although an abrupt onset and offset suggests reentry. Initial therapy for an atrial tachycardia involves rate slowing if necessary by blocking the AV node. More definitive therapy involve the use of an antiarrhythmic agent that has effects on atrial myocardium. This includes the class IA, IC, and III antiarrhythmic agents. ■

Core Case 43

A 70-year-old woman presents to her primary care physician with mild dyspnea and pre-syncope. The physician performs an ECG (ECG 43A) and a second ECG about 30 minutes later (ECG 43B).

ECG 43A

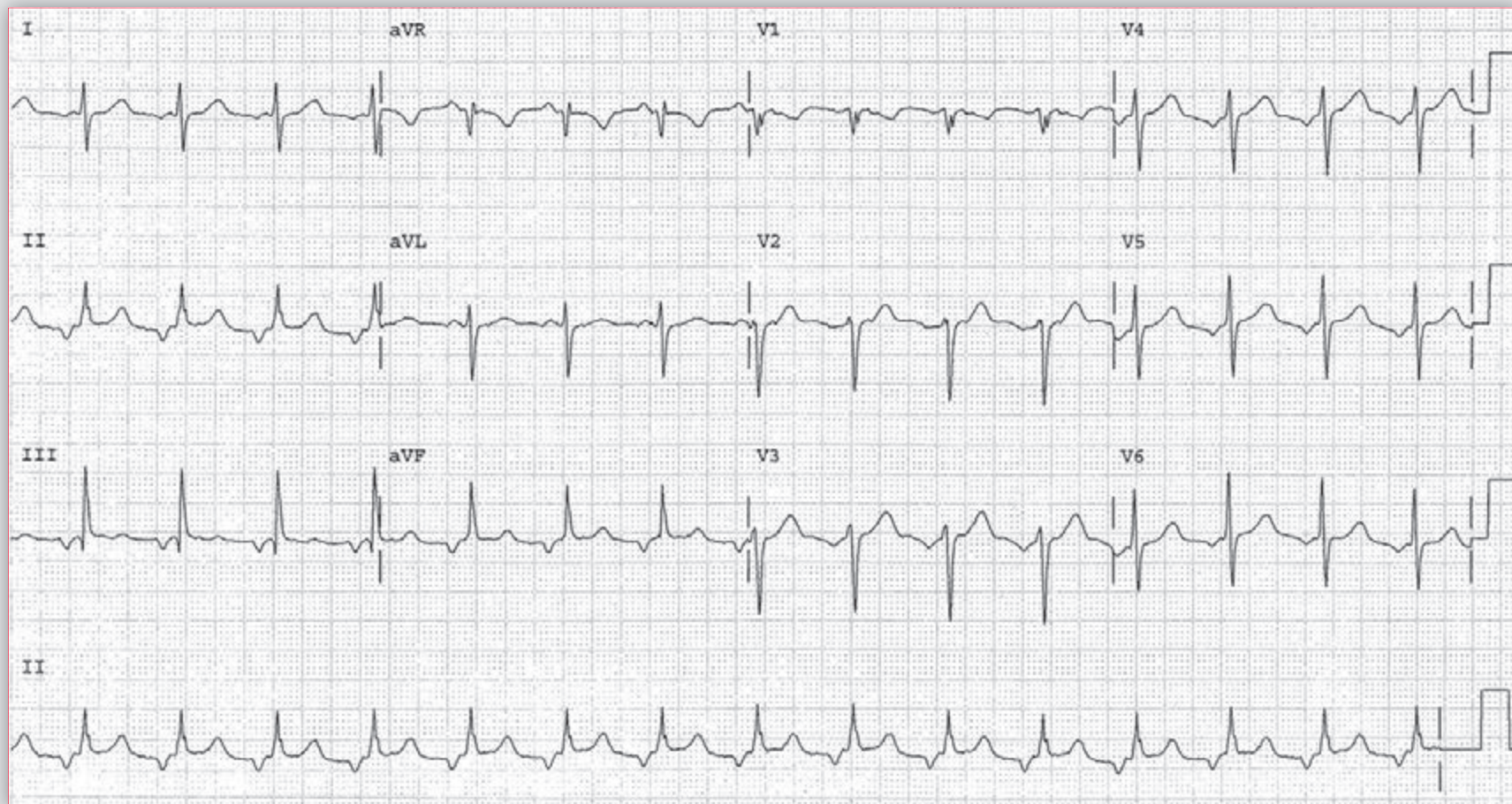


What does the ECG show?

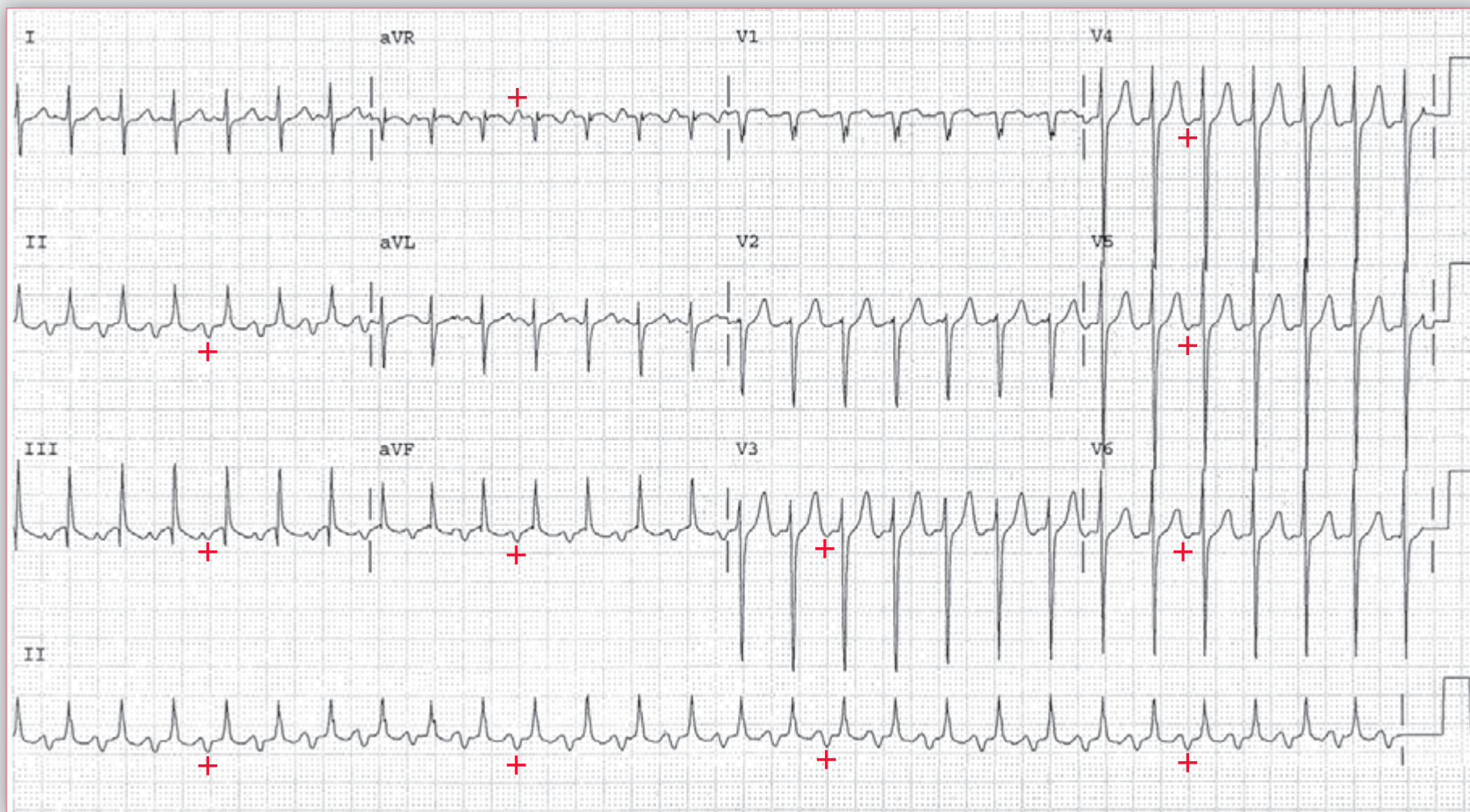
What is the likely diagnosis?

What therapy would be effective?

ECG 43B



Podrid's Real-World ECGs

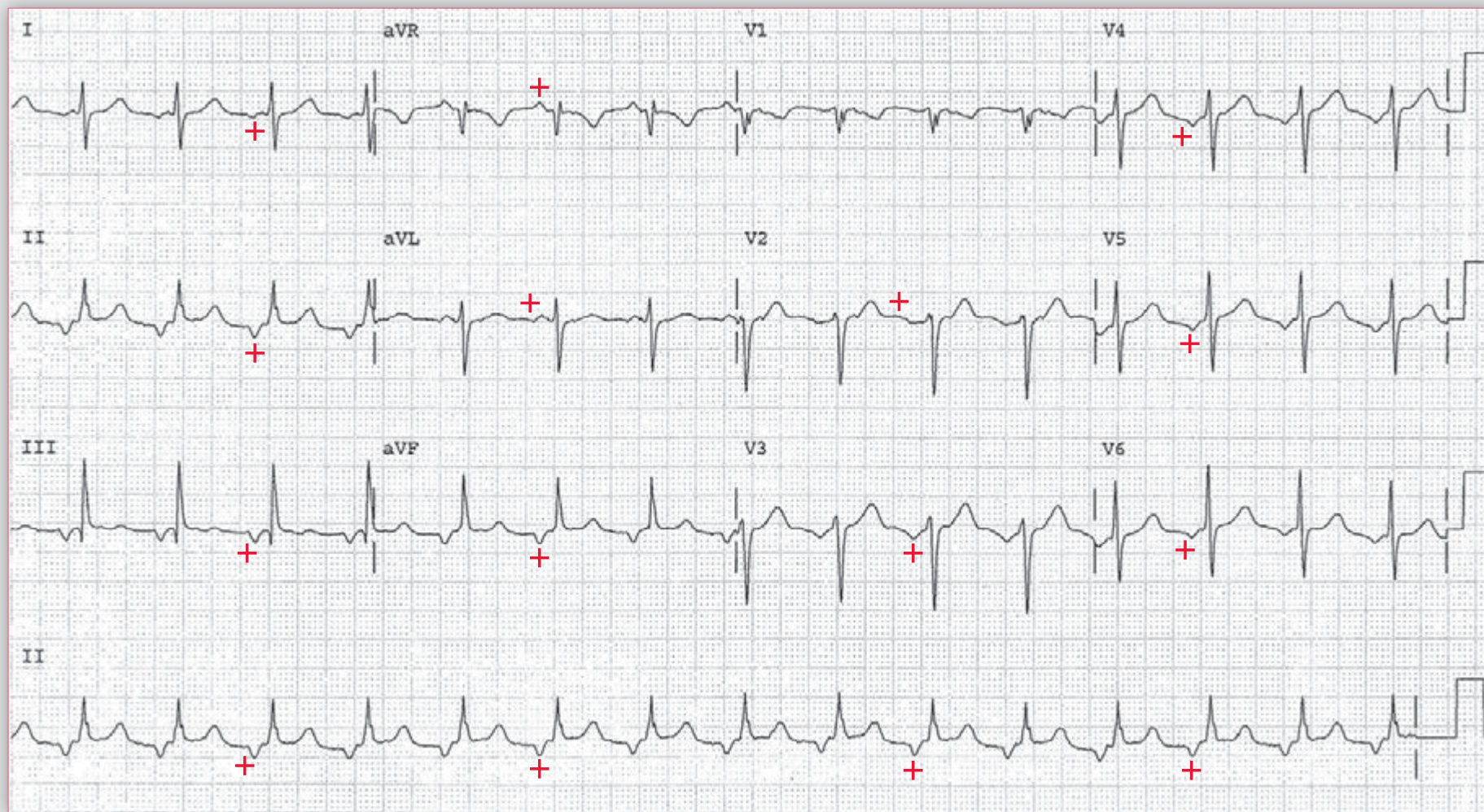


ECG 43A Analysis: Narrow complex supraventricular tachycardia (long-RP tachycardia), atrial tachycardia

ECG 43A shows a regular rhythm at a rate of 160 bpm. There is a P wave before each QRS complex (+) with a constant PR interval (0.18 sec). The P wave is negative in leads II, aVF, and V4–V6. The QRS complex has a normal duration (0.08 sec) and normal morphology. The axis is approximately $+90^\circ$ (QRS complex positive in lead aVF and biphasic in lead I). The QT/QTc intervals are normal (250/410 msec). This is a long-RP tachycardia (RP = 0.26 sec and

PR = 0.18 sec). Etiologies for a long-RP tachycardia include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atrioventricular reentrant tachycardia, and atypical or uncommon atrioventricular nodal reentrant tachycardia (fast-slow). As the P wave is negative in leads II and aVF, this is not a sinus tachycardia. A second atrial waveform is not seen, so it is not likely to be atrial flutter. *continues*

Podrid's Real-World ECGs



ECG 43B Analysis: Narrow complex rhythm, ectopic atrial rhythm

ECG 43B is from the same patient as ECG 43A. There is a regular rhythm at a rate of 90 bpm. The QRS complex duration, morphology, and axis are identical to what is seen in ECG 43A. The QT/QTc intervals are normal (380/440 msec). There is a P wave before each QRS complex (+) with a constant PR interval (0.18 sec). The P wave is negative in leads I, II, aVF, and V4–V6. Therefore, this is an atrial rhythm. The abnormal P wave has the same morphology as the P wave in ECG 43A and the PR intervals are the same in both ECGs. As the P-wave morphology is identical to that seen in ECG 43A, it is now obvious that the arrhythmia is an atrial tachycardia. As atrial tachycardia

is most often the result of an ectopic focus, the rate that this focus generates an impulse is variable, going from slow to fast depending upon multiple factors, including circulating catecholamines, which can accelerate the atrial focus, resulting in a tachycardia.

The treatment for an atrial tachycardia is first rate slowing, which is in this case might be achieved with β -blockade, given the two distinct atrial rates that are seen. More definitive therapy of the atrial arrhythmia itself involves the use of drugs that directly affect atrial tissue, which includes the class IA, IC, and III antiarrhythmic agents. ■

Core Case 44

An 84-year-old man is admitted for a bleeding gastric ulcer. He is noted to be tachycardic (ECG 44A) and at times his heart rate appears irregular on the telemetry monitor (ECG 44B).

ECG 44A

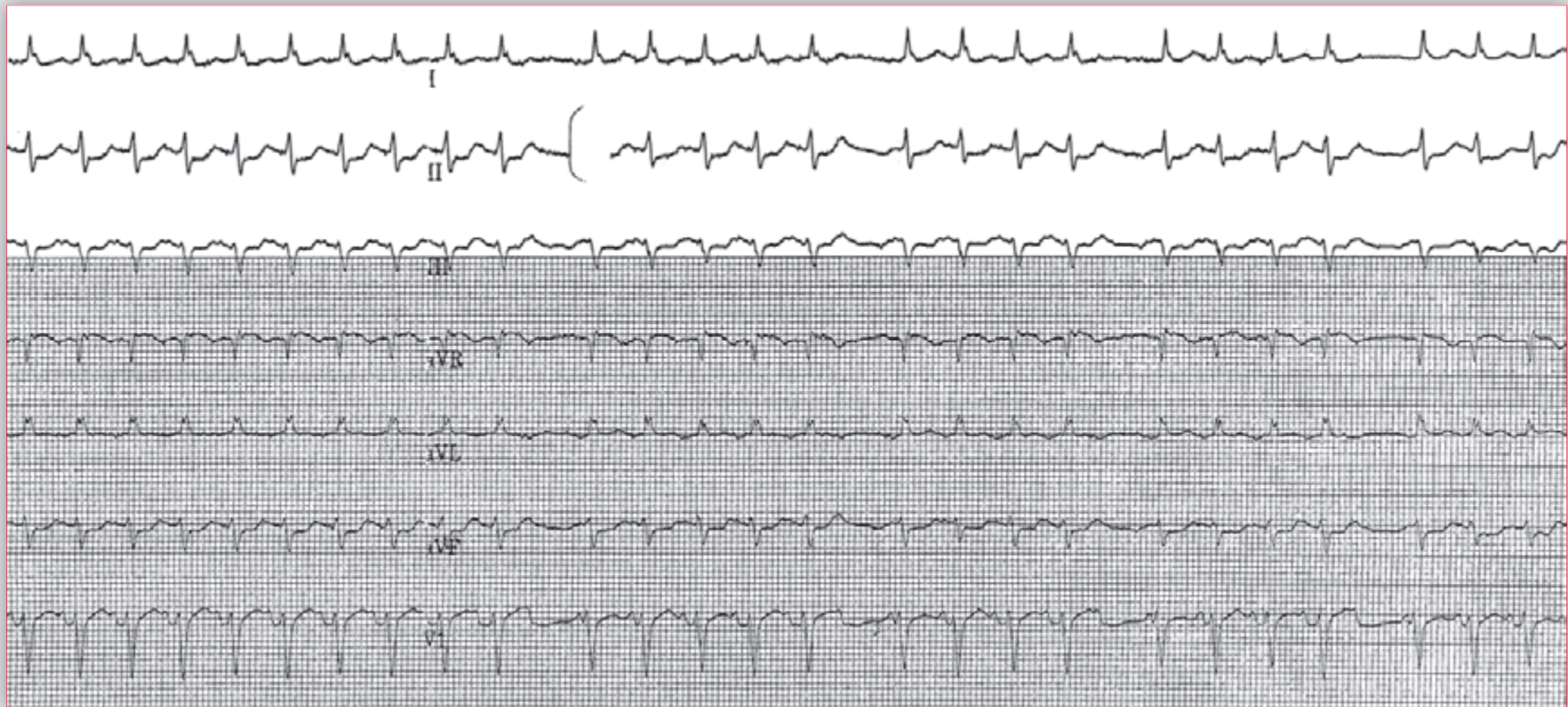


What do the ECGs show?

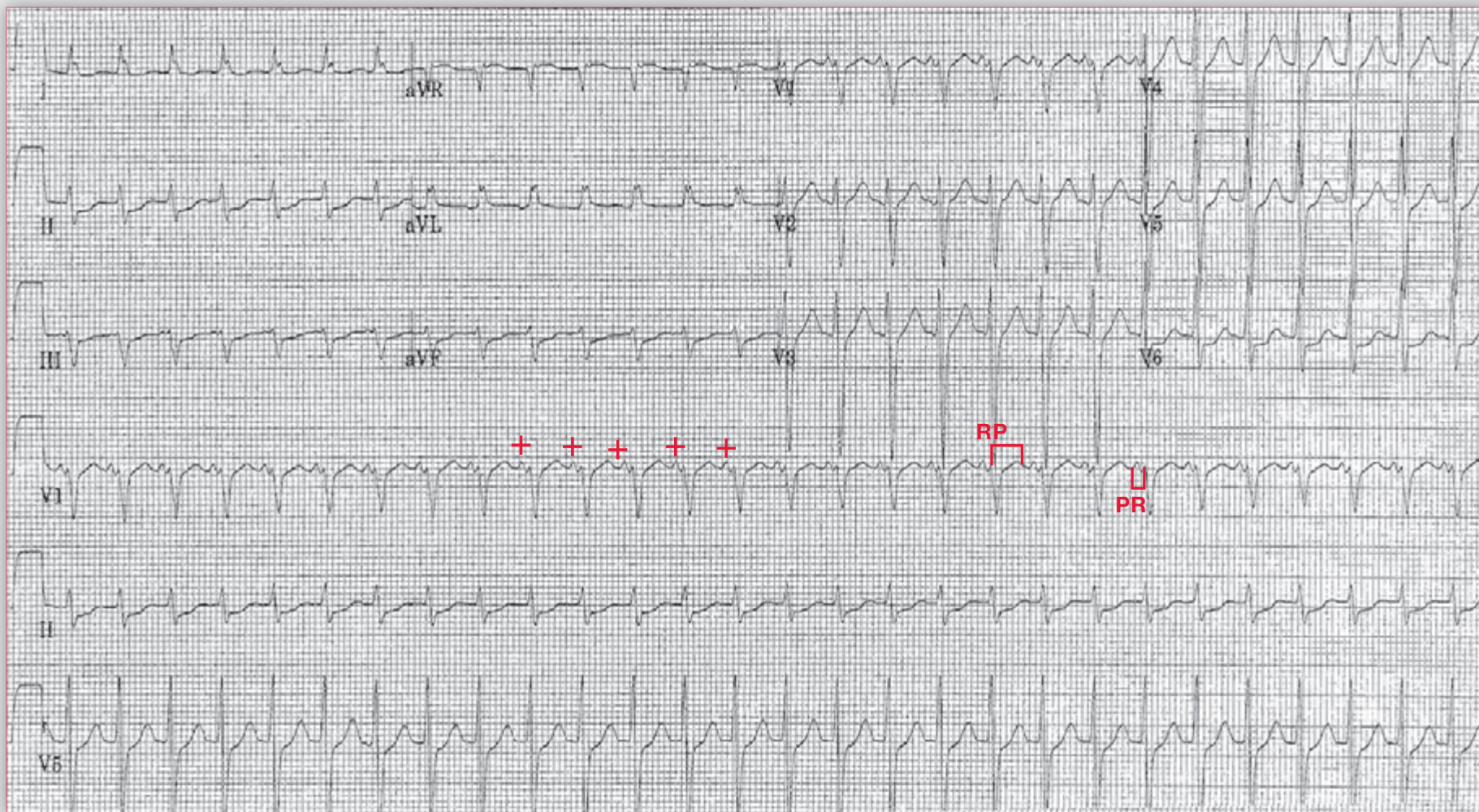
What is the likely diagnosis?

What treatment should be initiated?

ECG 44B



Podrid's Real-World ECGs



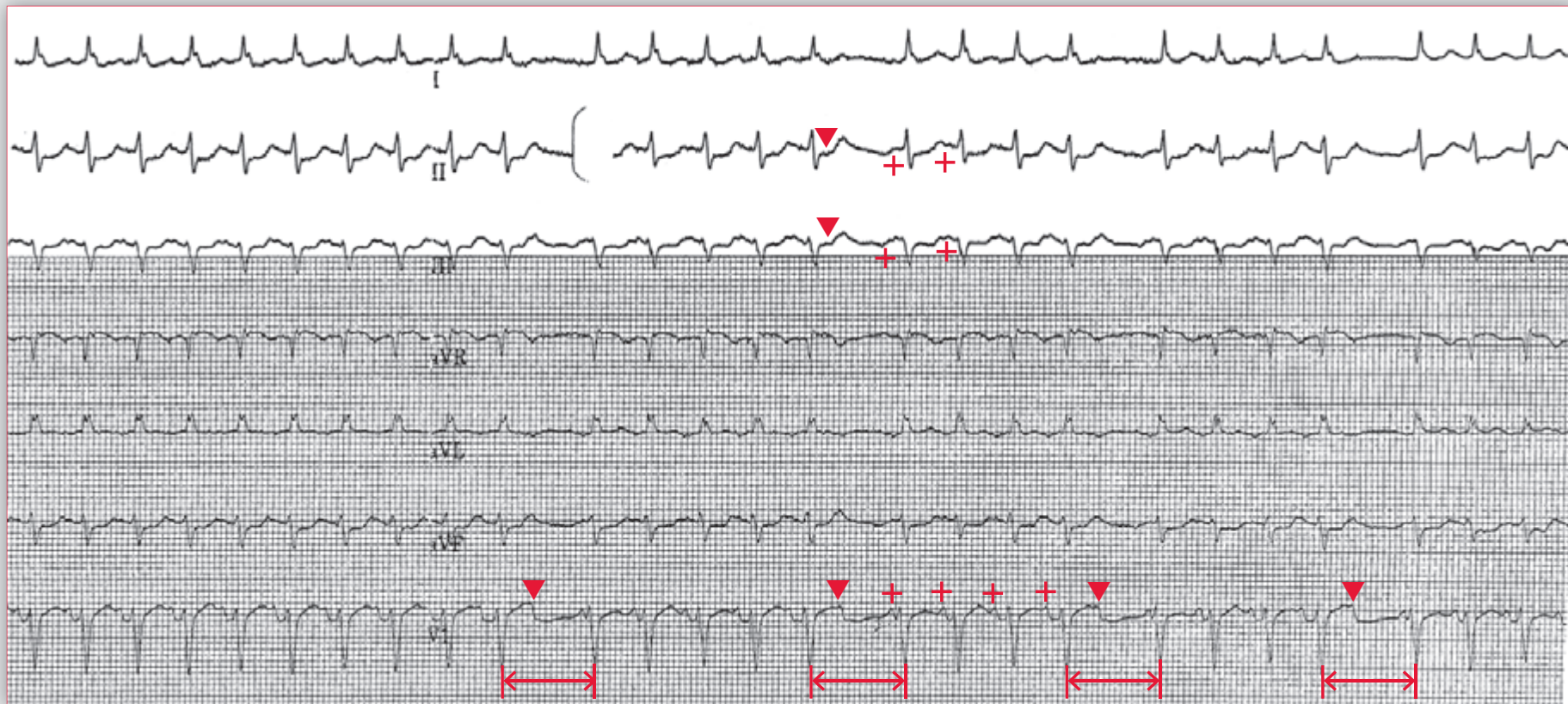
ECG 44A Analysis: Narrow complex supraventricular tachycardia, long-RP tachycardia (most probably atrial tachycardia), low-voltage limb leads, left axis

ECG 44A shows a regular rhythm at a rate of 180 bpm. The QRS complex duration is normal (0.08 sec), and it has a normal morphology, although there is low voltage in the limb leads (< 5 mm in each lead). The axis is physiologically leftward between 0° and -30° (positive QRS complex in leads I and II but negative in lead aVF). The QT/QTc intervals are normal (260/450 msec). The only obvious P waves (+) are seen in lead V1, and there is a long RP interval (0.26 sec) (\sqcap) and short PR

(0.10 sec) (\sqcup). Etiologies include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atypical or uncommon atrioventricular nodal reentrant tachycardia (fast-slow), or atrioventricular reentrant tachycardia. However, the actual etiology for this long-RP tachycardia cannot be established on this ECG. However, the most likely diagnosis is atrial tachycardia.

continues

Podrid's Real-World ECGs



ECG 44B Analysis: Narrow complex supraventricular tachycardia, long-RP tachycardia atrial tachycardia with occasional 2:1 AV block due to Mobitz type I second-degree AV block (Wenckebach)

ECG 44B is a regularly irregular rhythm as a result of several long RR intervals (\leftrightarrow), all of which are the same (0.68 sec or rate 90). The short RR intervals have a rate of 180 bpm, which is identical to the ventricular rate seen in ECG 44A. As on ECG 44B, P waves (+) can be seen in lead V1, and the long RR intervals are the result of a nonconducted P wave (\blacktriangledown), or intermittent second-degree AV block, defined as an occasional non-conducted P wave. The atrial rate is 180 bpm, and when two sequential P waves are present during the long RR interval, it can be seen that the P waves are distinct with an isoelectric baseline between them. In addition, the P waves are negative in leads II and

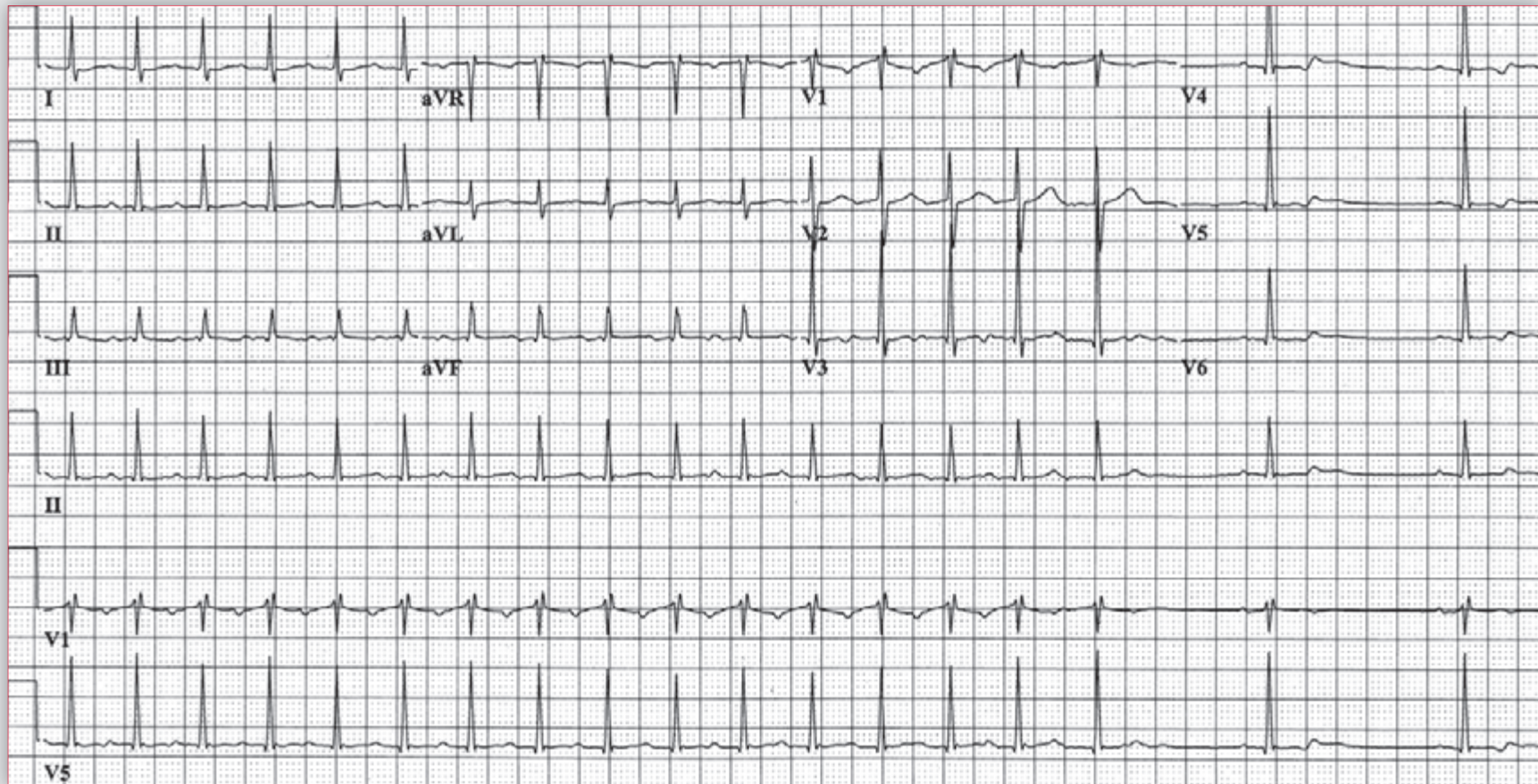
aVF. Hence the rhythm in ECG 44A is atrial tachycardia with 1:1 AV conduction, while ECG 44B shows atrial tachycardia with 1:1 conduction and intermittent 2:1 AV conduction. It can be seen that prior to the nonconducted P wave, there is a gradual lengthening of the PR interval, indicative of Mobitz type I second-degree AV block or Wenckebach.

Initial therapy is to slow the ventricular rate by increasing AV block using an AV nodal blocking agent, such as a β -blocker, calcium-channel blocker, or digoxin. Definitive therapy for the arrhythmia requires the use of a standard antiarrhythmic agent, *ie*, class IA, IC, or III. ■

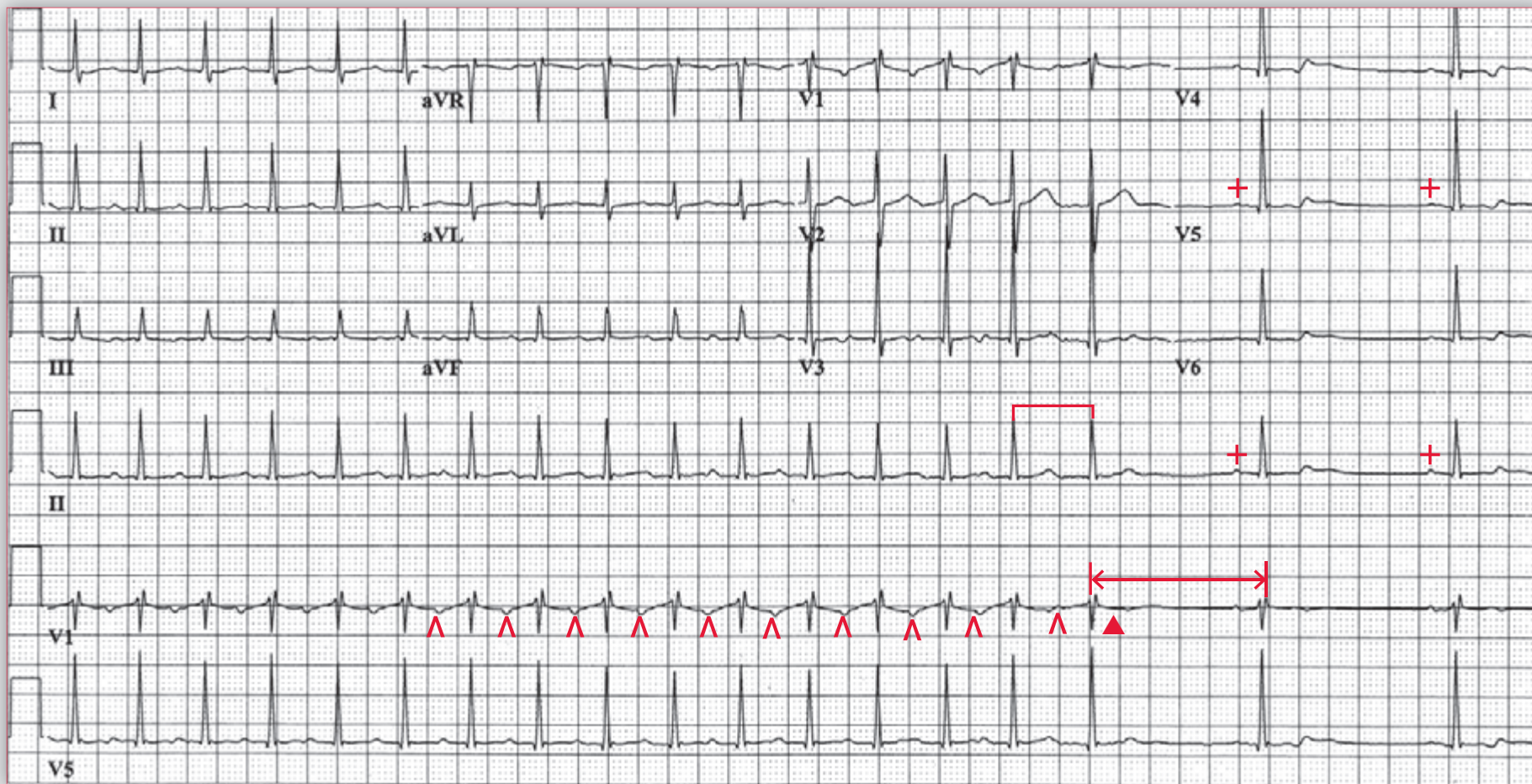
Notes

A 40-year-old woman has periods of tachycardia noted during an admission for alcohol withdrawal. The arrhythmia breaks abruptly, as is shown in this ECG.

What is the likely diagnosis?



Podrid's Real-World ECGs



ECG 45 Analysis: Narrow, complex supraventricular tachycardia, long-RP tachycardia, atrial tachycardia, termination to sinus bradycardia

ECG 45 shows a regular rhythm at a rate of 136 bpm. The QRS complex duration is normal (0.08 sec) and the morphology is normal. The axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The RR interval between the fifteenth and sixteenth QRS complex is slightly longer (\square) than the other RR intervals, and after the sixteenth QRS complex there is an abrupt termination of the tachycardia (\leftrightarrow). After the termination, the last two QRS complexes, which have the same duration and morphology as the first 16 complexes, are at a slow rate (46 bpm). These last two QRS complexes have a P wave before them (+) with a stable PR interval (0.20 sec). The P wave is positive in leads II and V5 and are likely sinus. Hence this is a sinus bradycardia. The QT/QTc intervals are normal (320/450 msec). Although there is no distinct P wave seen during the initial tachycardia, by comparing the QRS complex and ST segments with those of the two sinus complexes, it can be seen that there is a negative waveform (\wedge) at the beginning of or during the T wave of each QRS complex. This waveform is not present in the sinus complexes. Hence this is a P wave

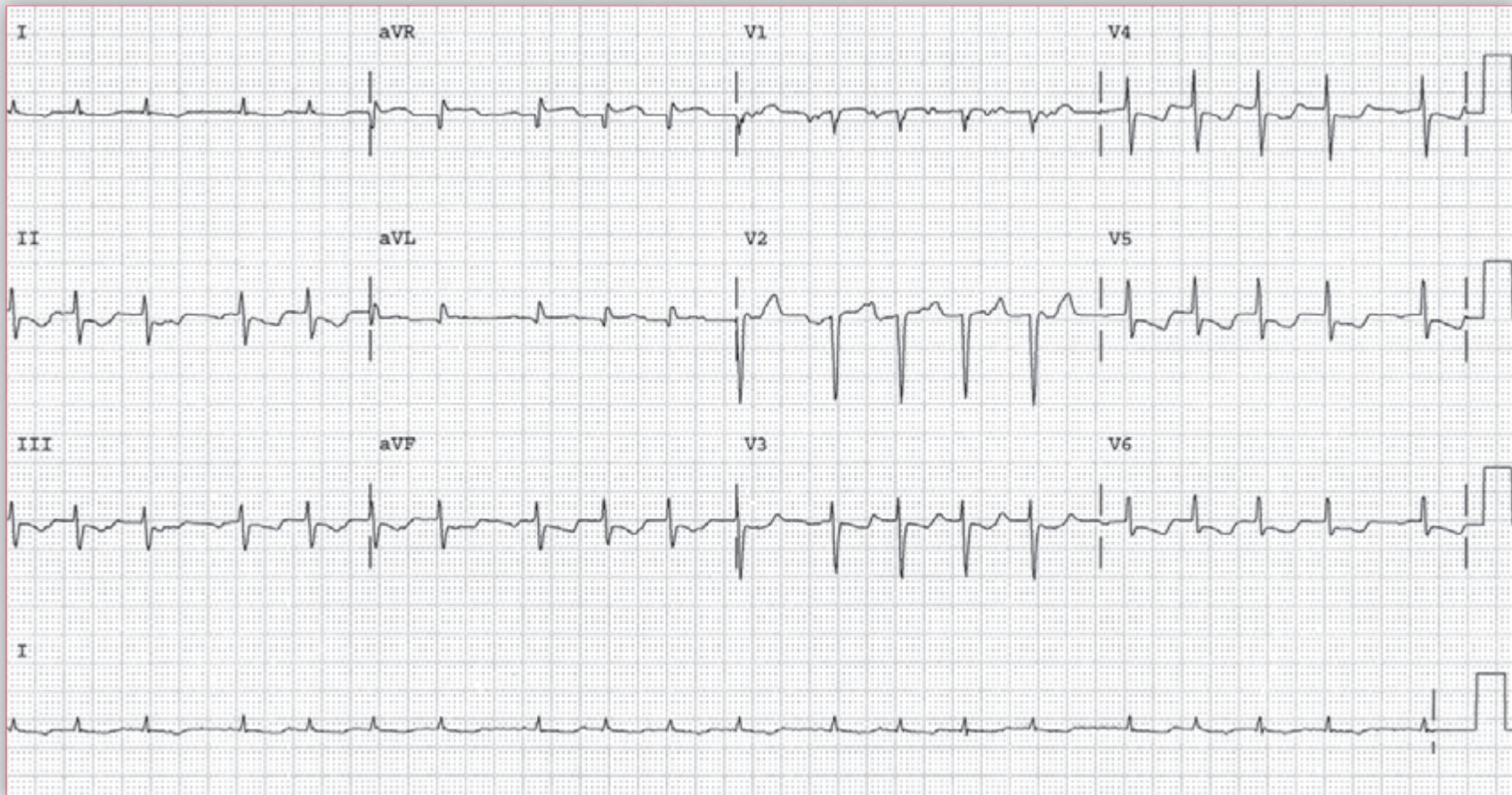
and the RP interval (0.28 sec) is slightly longer than the PR interval (0.24 sec). Etiologies for a long-RP tachycardia include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, an ectopic junctional tachycardia, atypical atrioventricular nodal reentrant tachycardia and an atrioventricular reentrant tachycardia. The etiology is not likely sinus tachycardia, as the PR interval during the tachycardia is longer than the PR interval with the sinus bradycardia. Atrial flutter is not likely, as there is no evidence for a second atrial waveform. A reentrant arrhythmia is less likely because of the slight lengthening of the RR interval just prior to termination. This is not usually seen with a reentrant arrhythmia in which there is fixed circuit and a fixed time for the impulse to travel around this circuit. In addition, the arrhythmia terminates with the absence of a P wave, *ie*, there is no P wave after the last QRS complex of the tachycardia (\blacktriangle). This is the mode of termination of atrial arrhythmia, *ie*, the atrial focus stops firing and the arrhythmia terminates. Therefore, this is an atrial tachycardia. ■

Notes

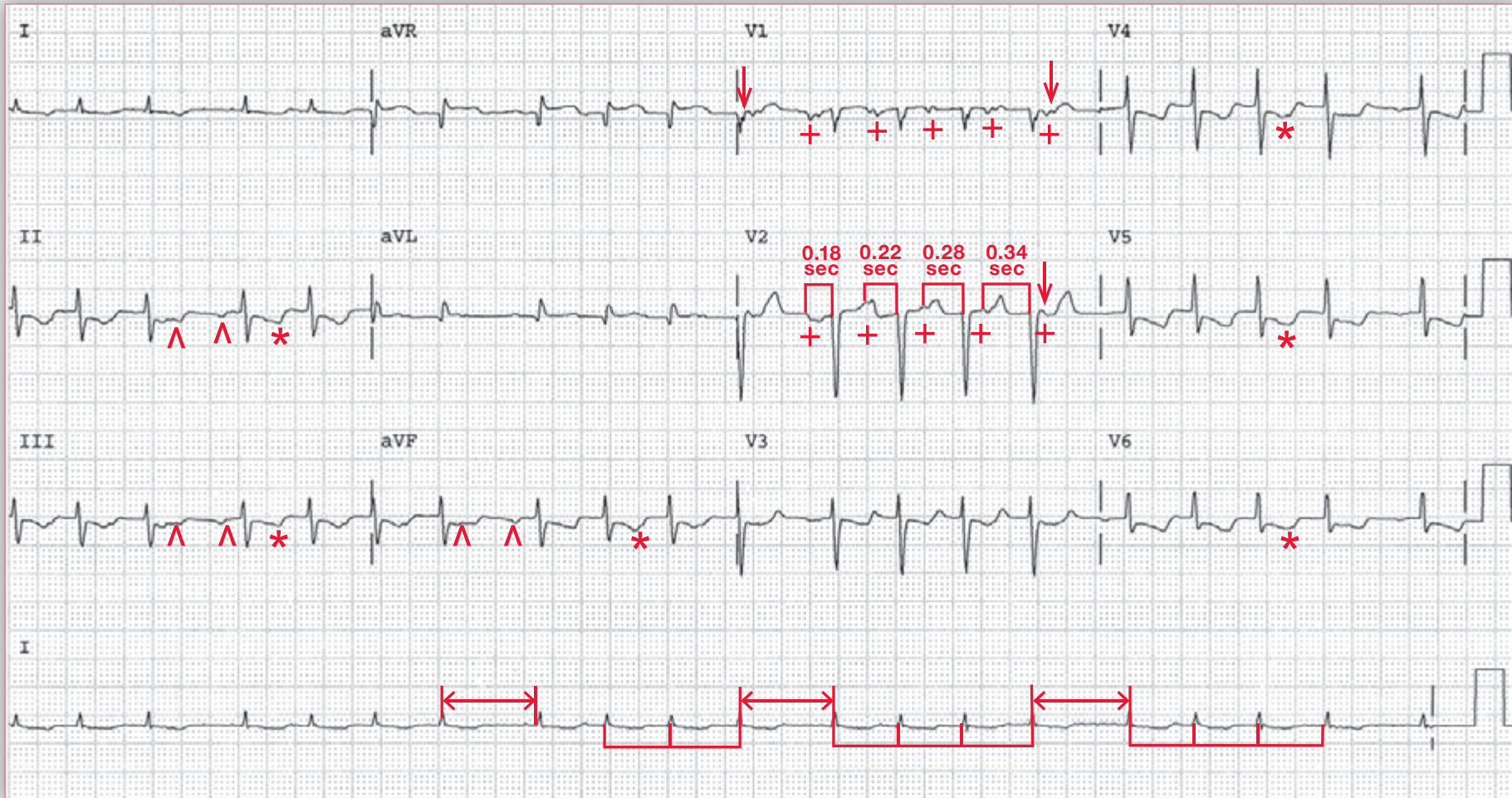
A 42-year-old woman has recurrent palpitations. She takes her own pulse and notes that her heart is rapid and irregular. She presents to a walk-in unit and an ECG is performed.

What does the ECG show?

What would be the best therapy?



Podrid's Real-World ECGs



ECG 46 Analysis: Atrial tachycardia with Mobitz type I second-degree AV block, 5:4 Wenckebach, nonspecific ST-T wave changes

ECG 46 shows the rhythm is regularly irregular, and there is evidence of group beating. All the short RR intervals (\sqcup) are the same, with an average rate of 130 bpm, while all the long intervals (\leftrightarrow) are the same, at a rate of 94 bpm. The average rate is 120 bpm. There are P waves (+) seen, primarily in leads V1–V2. The atrial rate is 142. On close inspection negative P waves (^) can be seen in leads II, III, and aVF, especially after the pause. Hence this is an atrial tachycardia. As can be seen in leads V1–V2, there is progressive lengthening of the PR interval, from 0.18 second to 0.34 second (\sqcap), while the fifth P wave (\downarrow) is not conducted. Hence this is an atrial tachycardia with 5:4 Wenckebach (Mobitz type I second-degree AV block).

The QRS complex duration is normal (0.08 sec) and there is a normal morphology. The axis is 0° as the QRS complex in lead aVF is biphasic. The QT/QTc intervals are normal (320/450 msec). There are nonspecific ST-T wave changes seen in leads II, aVF, and V3–V6 (*).

Mobitz type I second-degree AV block or Wenckebach is due to decremental conduction through the AV node, resulting in progressive lengthening of the PR interval culminating with one nonconducted P wave after which the PR interval shortens to the baseline value. Wenckebach can be seen with sinus rhythm, atrial tachycardia and atrial flutter. In this patient, appropriate therapy would be to further slow conduction through the AV node, which would perhaps result in a shorter Wenckebach cycle length, for example 3:2, or perhaps 2:1 AV block. ■

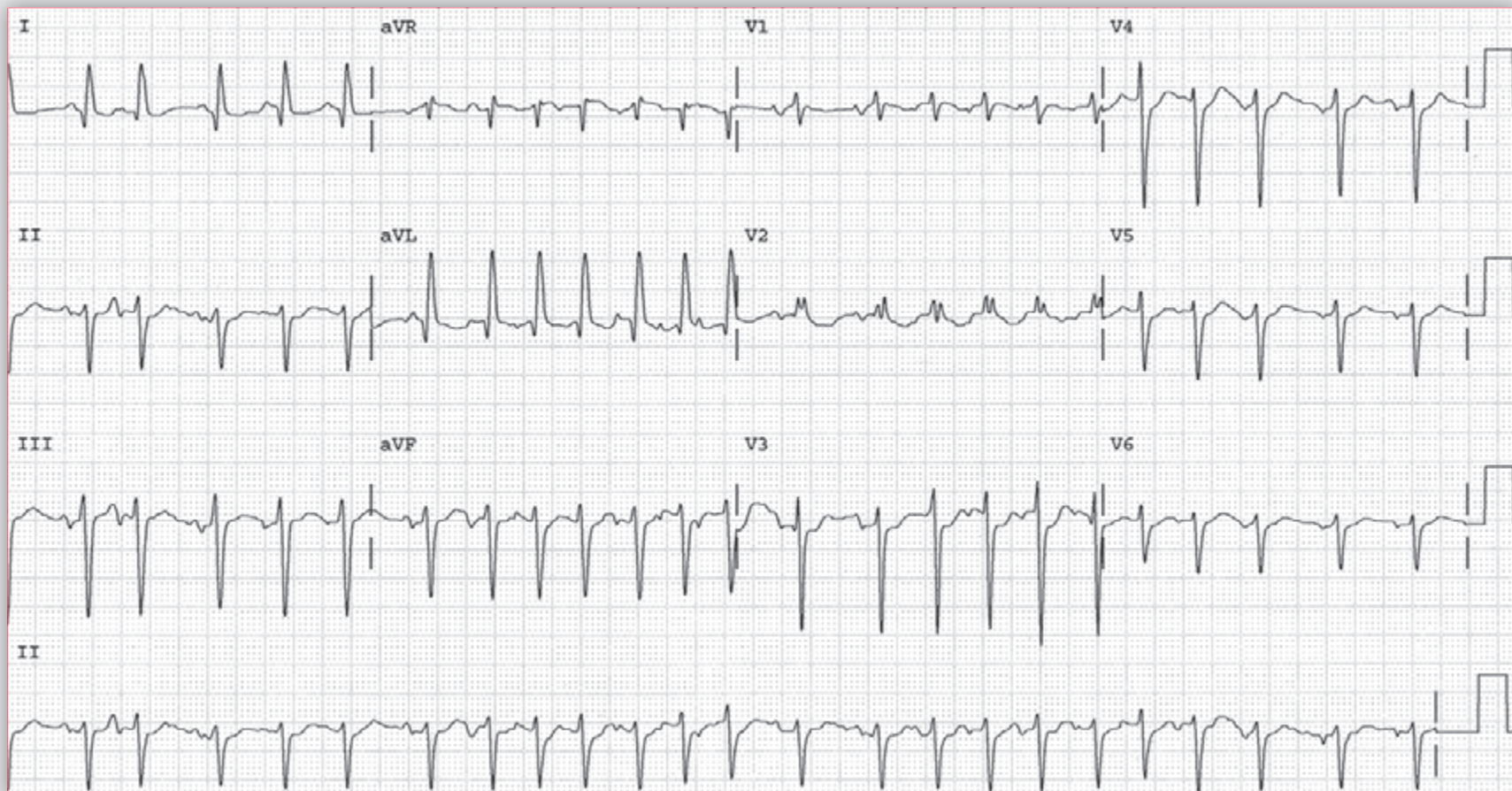
Notes

A 58-year-old man with severe emphysema/COPD is admitted with progressively worsening dyspnea. On presentation, it is felt that he has a COPD exacerbation. He is noted to have a rapid heart rate.

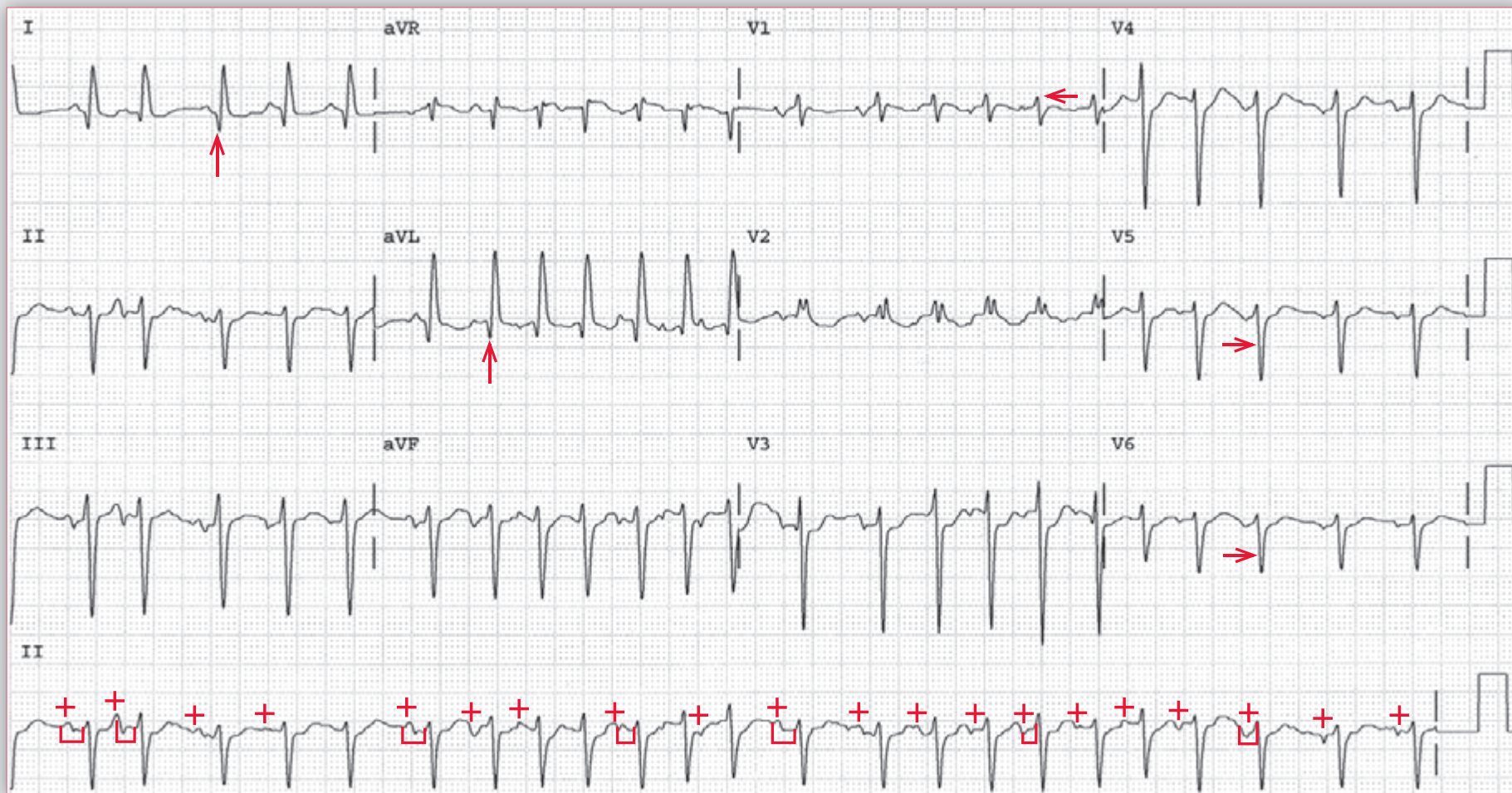
What does the ECG show?

What is the likely etiology of his arrhythmia?

What therapy is important?



Podrid's Real-World ECGs



ECG 47 Analysis: Multifocal atrial tachycardia, left anterior fascicular block, old lateral infarction, right ventricular hypertrophy

ECG 47 shows the rhythm is irregularly irregular as there is no pattern to the RR intervals seen. The average heart rate is 144 bpm. The QRS complex duration is normal (0.10 sec) and the axis is extremely leftward between -30° and -90° (positive in lead I and negative in leads II and aVF). As the QRS complexes in leads II and aVF have an rS morphology, this is a left anterior fascicular block. Noted are Q waves in leads I and aVL (\uparrow), consistent with an old lateral wall myocardial infarction. There are prominent R waves in V1 (\leftarrow), and poor R wave progression from leads V3–V6. In V5–V6, the S wave is deep (\rightarrow) with an R/S ratio < 1 . These features suggest right ventricular hypertrophy.

There are only three arrhythmias associated with an irregularly irregular rhythm. This includes sinus arrhythmia (one P-wave morphology and a stable PR interval); multifocal atrial tachycardia (rate > 100 bpm), or multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm) (> 3 different P-wave morphologies with no P-wave morphology being dominant and variable PR intervals); and atrial fibrillation (no organized P waves). In this ECG, there are

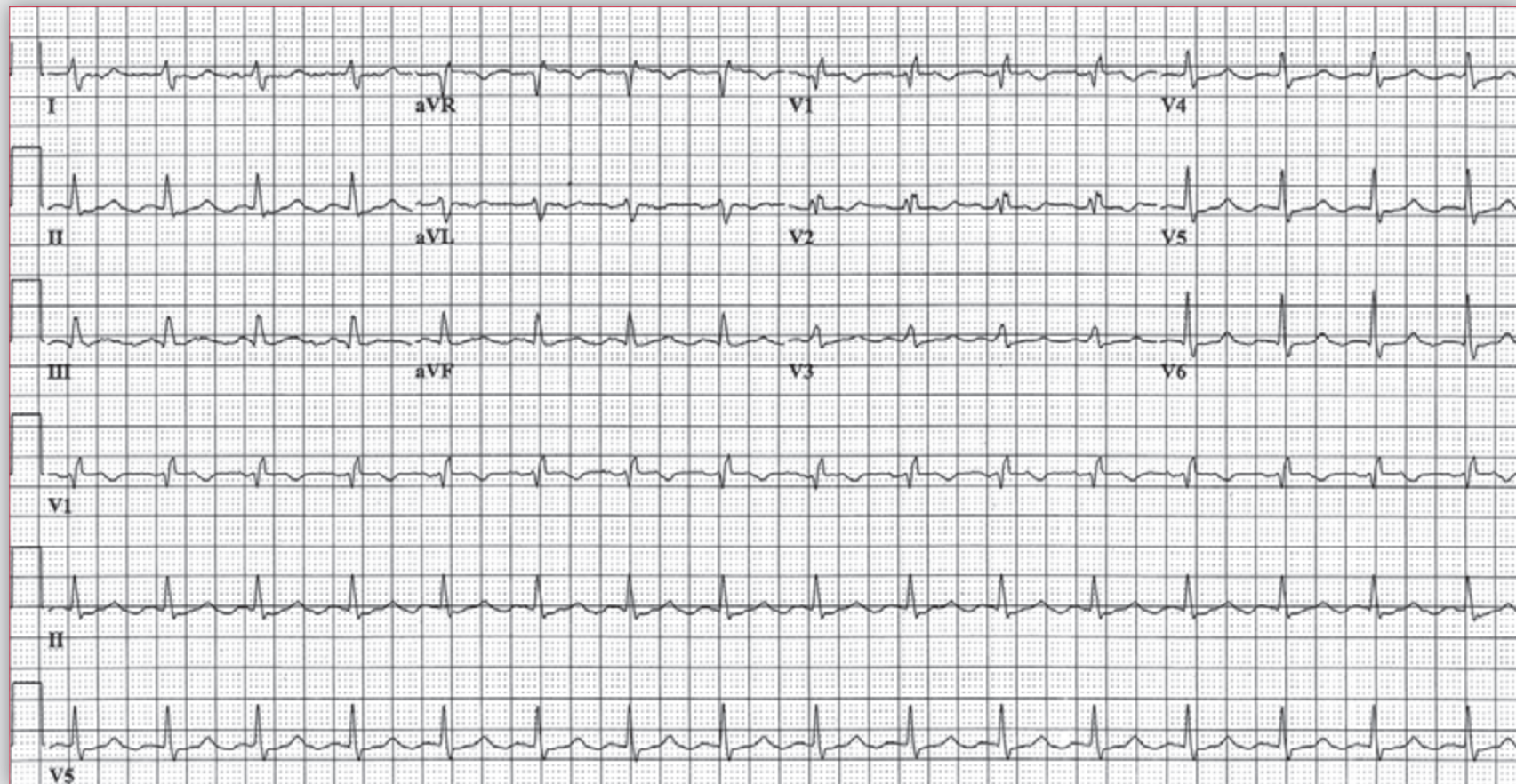
P waves (+) seen before each QRS complex with variable or nonconsistent PR intervals (\sqcup). In addition, the P-wave morphology is variable, with > 3 different morphologies seen. These features (irregularly irregular rhythm at a rate > 100 , variable P-wave morphologies and variable PR intervals) are the features of a multifocal atrial tachycardia.

Multifocal atrial tachycardia is most commonly associated with pulmonary disease, especially if there is pulmonary artery hypertension with elevated right atrial and right ventricular pressures. It may also be seen with severe left ventricular dysfunction and heart failure due to any cause, *ie*, valvular, cardiomyopathy, or coronary disease. As the mechanism for this arrhythmia is multiple ectopic foci, this arrhythmia cannot be terminated with cardioversion. Therapy is primarily directed at rate control by blocking the AV node, and the most effective agents are β -blockers or a calcium-channel blocker, especially verapamil. Although data are poor, there is some suggestion that therapy with magnesium and/or potassium may be beneficial. ■

Core Case 48

A 72-year-old man with a history of COPD is admitted to the hospital because of fever, a productive cough, and worsened shortness of breath. The symptoms have become progressively worse over the past week. A chest x-ray demonstrates a right middle lobe infiltrate and the WBC count is 18,000. A diagnosis of a community

ECG 48A



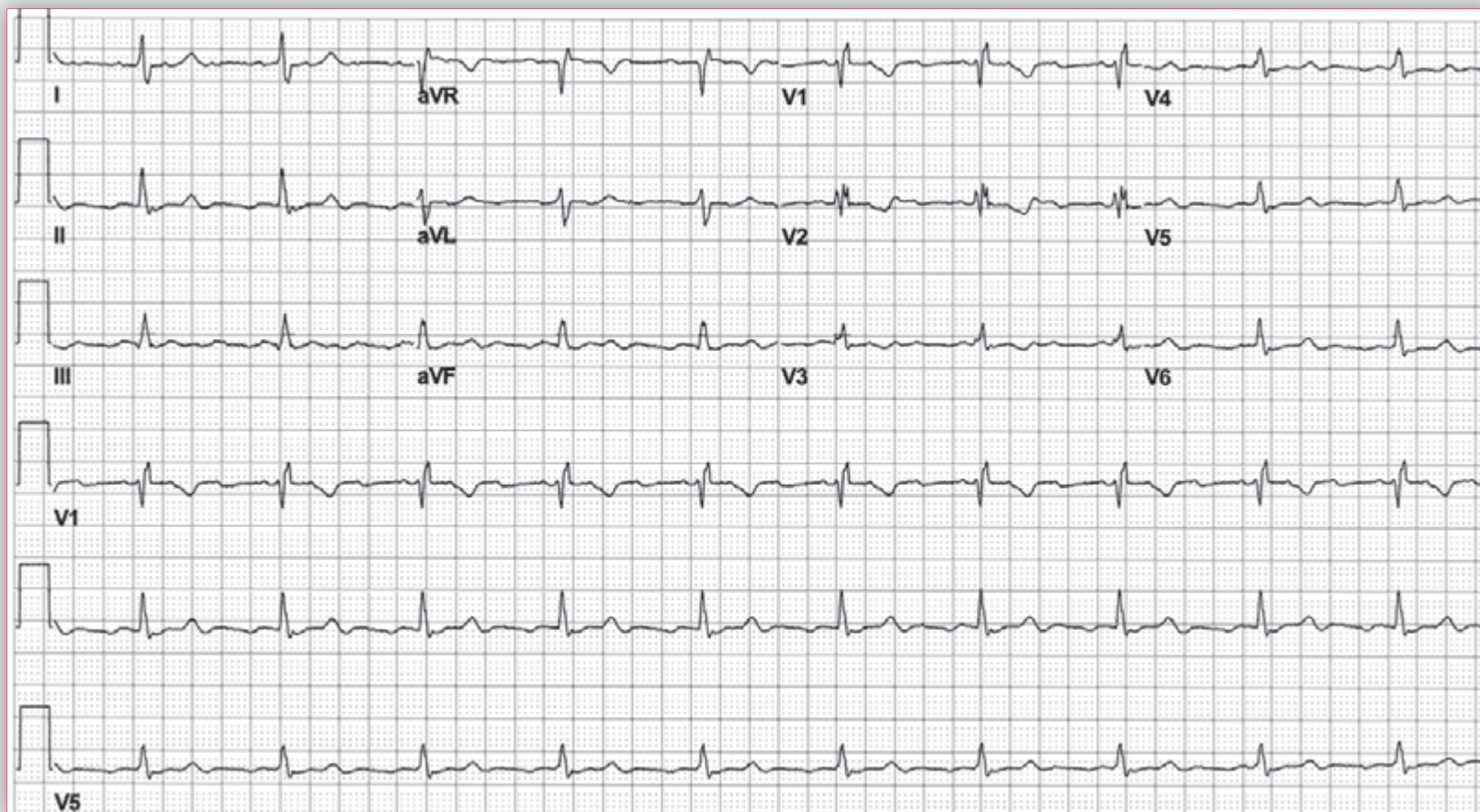
acquired pneumonia is made. A routine ECG is obtained (ECG 48A) and felt to be normal. On the following day, after therapy with fluids and antibiotics are begun, another routine ECG is obtained (ECG 48B), and this causes some concern. Consultation from a cardiologist is obtained and the two ECGs are compared.

What is the ECG abnormality of concern?

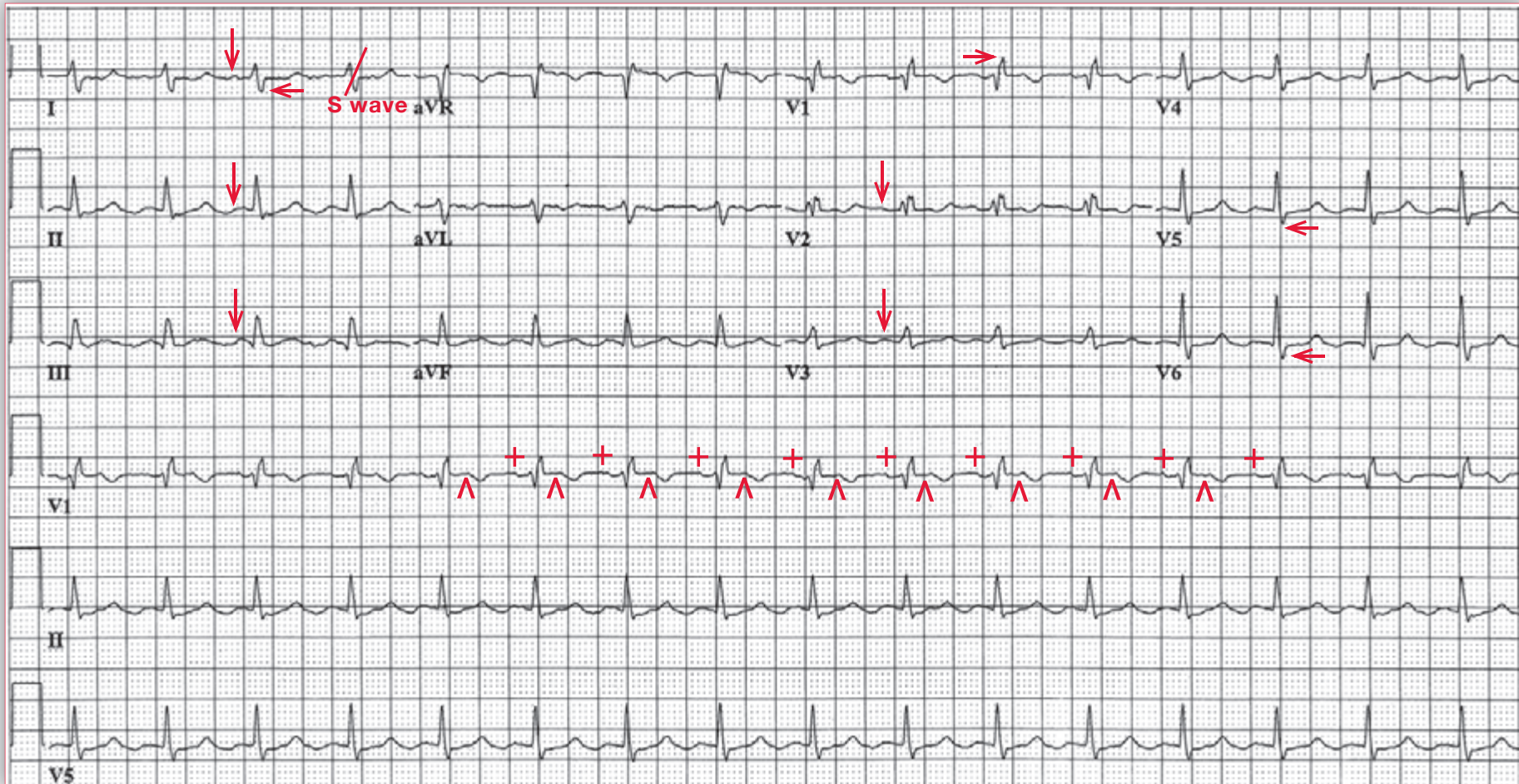
What rhythm is seen on ECG 48A and ECG 48B?

Is any additional therapy warranted?

ECG 48B



Podrid's Real-World ECGs



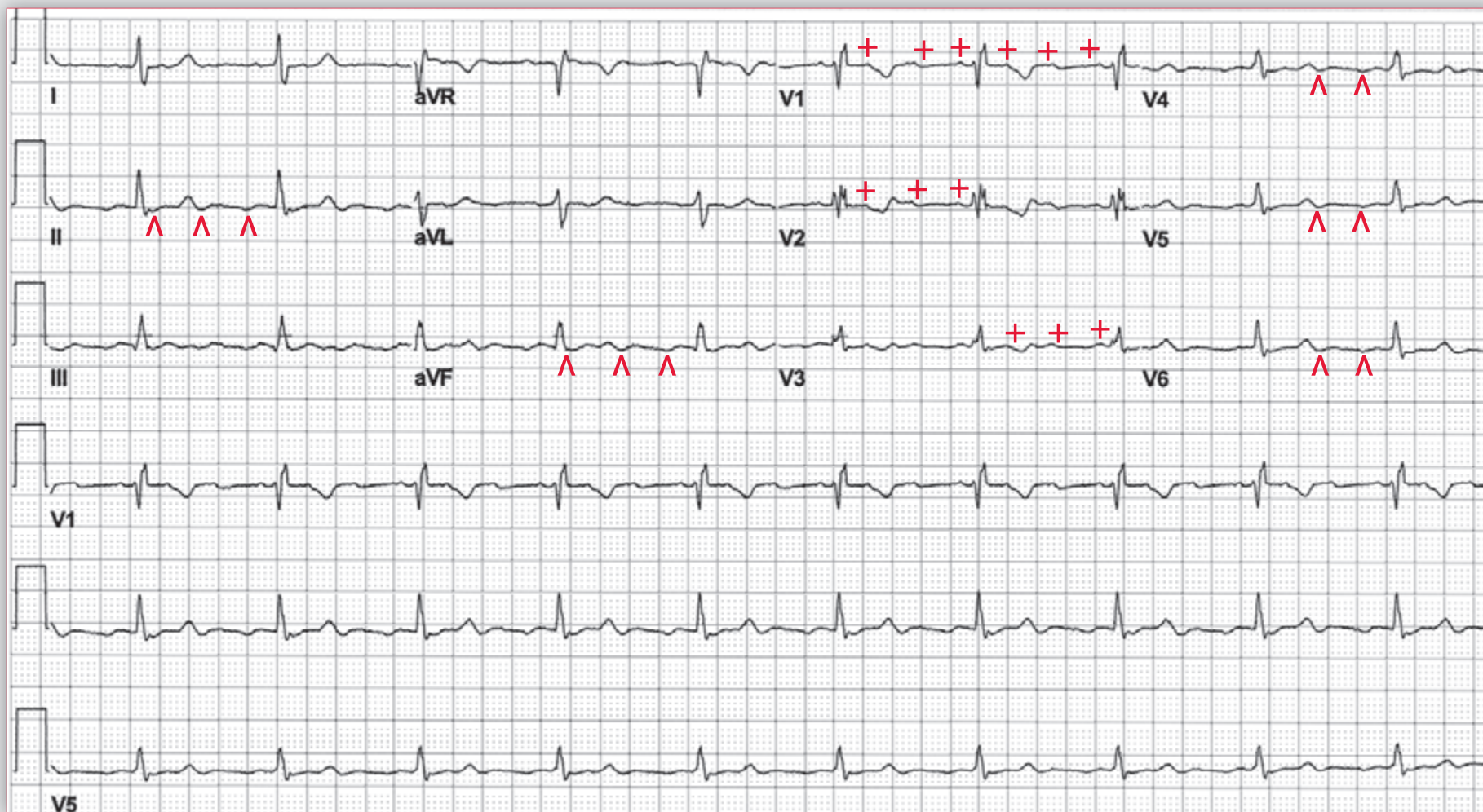
ECG 48A Analysis: Narrow complex supraventricular tachycardia, right ventricular conduction delay, atrial tachycardia with 2:1 AV block, low QRS complex voltage

ECG 48A shows a regular rhythm at a rate of 100 bpm. The QRS complex duration is slightly prolonged (0.11 sec), and the morphology is that of a right bundle branch block with an RSR' in V1 (→) and broad S wave in leads I and V5–V6 (←). As the QRS complex is not quite 0.12 second, this is an intraventricular conduction delay to the right ventricle (often called an incomplete right bundle branch block). The axis is normal between 0° and +90° (positive QRS complex in leads I when ignoring the terminal S wave, which represents delayed right ventricular depolarization, and aVF). The QT/QTc intervals are normal (320/410 msec). There is low QRS complex voltage, defined as an amplitude < 5 mm in each limb lead and < 10 mm in each precordial

lead. There are no obvious P waves seen. However, in lead V1 there are positive waveforms seen. The first is just before the QRS complex (+), and this is simultaneous with positive waveforms seen in leads V2–V3 as well as leads I, II, and III (↓). With careful inspection a second P wave can be identified; it is superimposed on the ST segment just before the T wave in lead V1 (^). These are atrial waveforms occurring with regular intervals and the atrial rate is 200 bpm. This indicates that the rhythm is an atrial tachycardia with 2:1 AV block. It is possible that it could be atrial flutter (which is slower than normal) with 2:1 AV block, although the presence of distinct P waves supports a diagnosis of atrial tachycardia.

continues

Podrid's Real-World ECGs



ECG 48B Analysis: Atrial tachycardia with 3:1 AV block

ECG 48B is from the same patient as ECG 48A. There is a regular rhythm at a rate of 62 bpm. The QRS duration, morphology, and axis are identical to ECG 48A. The QT/QTc intervals are also normal and the same as seen in ECG 48A. As a result of the slower rate, distinct P waveforms can now be seen in leads V1–V3 (+). The atrial rate is 200 bpm and is identical to the atrial rate in ECG 48A. Closer inspection of leads II, III, aVF, and V4–V6 reveals that the atrial waveforms are negative (^). Hence this is not a sinus tachycardia. Although the most likely diagnosis is an atrial tachycardia with 3:1 AV block, it is possible that the arrhythmia is atrial flutter with 3:1 block, although the atrial rate is slow for atrial flutter, which usually presents with an atrial rate of 260 to 320 bpm. However, the atrial rate can be slower if there is atrial disease or antiarrhythmic drugs are being used. However,

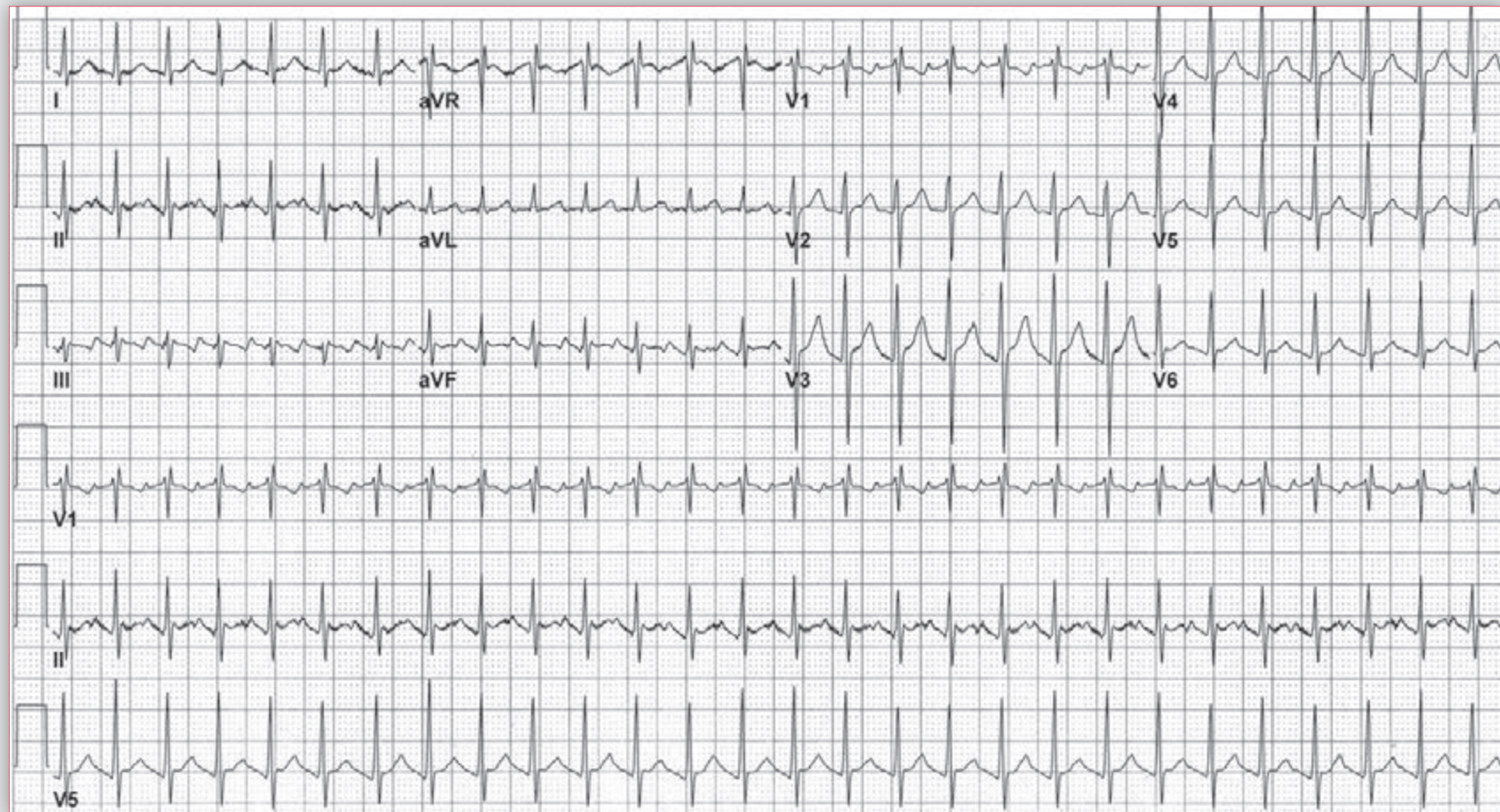
even if the rate is slower, atrial flutter maintains the usual flutter morphology, *ie*, continuously undulating baseline without an isoelectric baseline between each flutter waves. In contrast, atrial tachycardia has distinct P waves with an isoelectric baseline in between each P wave. This is the morphology seen in this ECG. Therefore, this is an atrial tachycardia with 2:1 AV conduction (ECG 48A) and 3:1 AV conduction (ECG 48B).

Although the patient has an atrial tachycardia, the ventricular response rate is slow and hence there is no clinical reason for additional therapy. It is possible that the atrial tachycardia will revert after adequate therapy of pneumonia. If the atrial tachycardia persists, therapy with a class IA, class IC, or class III antiarrhythmic drug can be considered. ■

Core Case 49

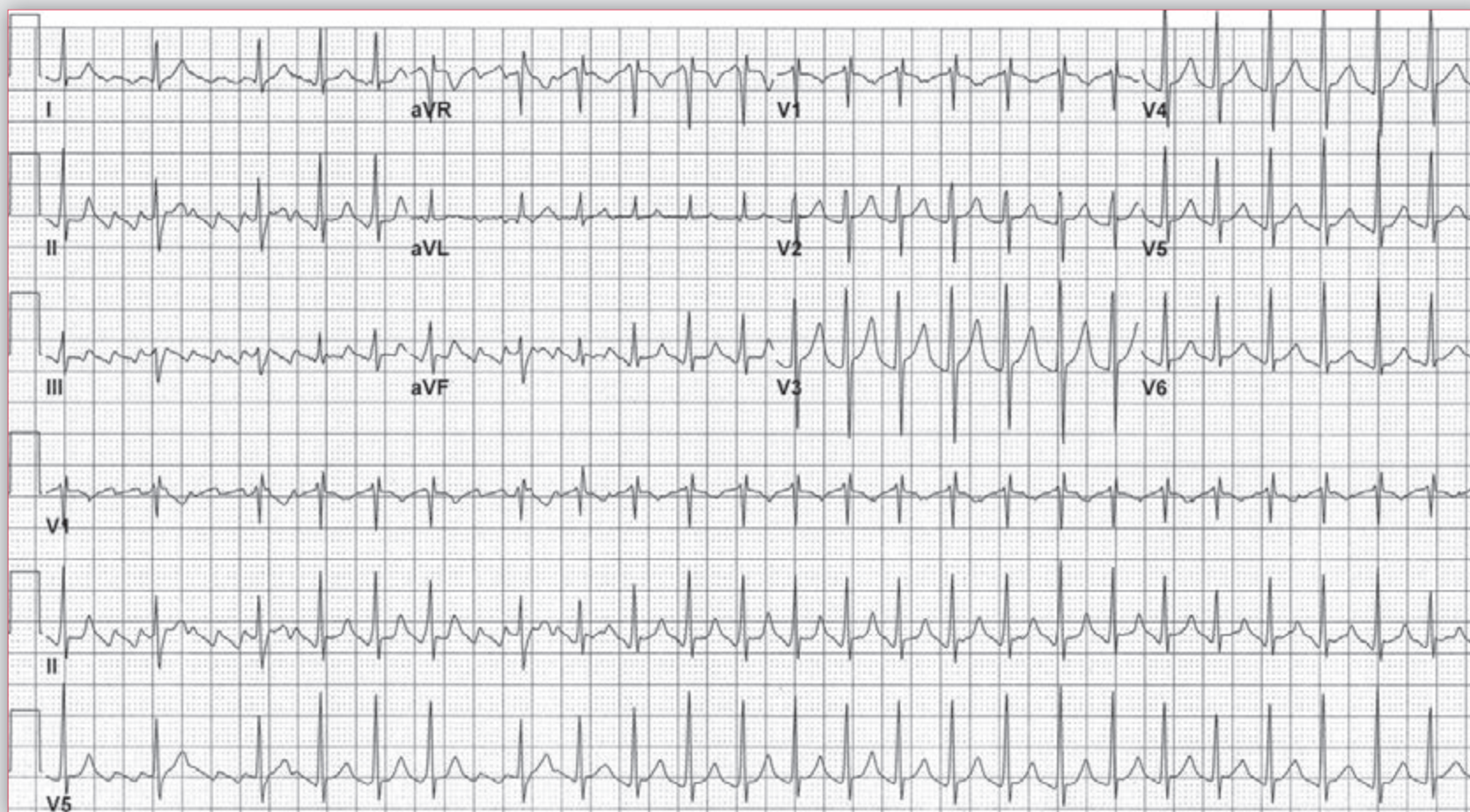
A 51-year-old man has a history of palpitations that occur every few months. He is in his physician's office for a routine physical examination. While being examined, his heart rate abruptly increases and he complains of the typical palpitations. An ECG is obtained (ECG 49A). After several minutes, his heart rate slows, but becomes irregular. Another ECG is obtained (ECG 49B).

ECG 49A

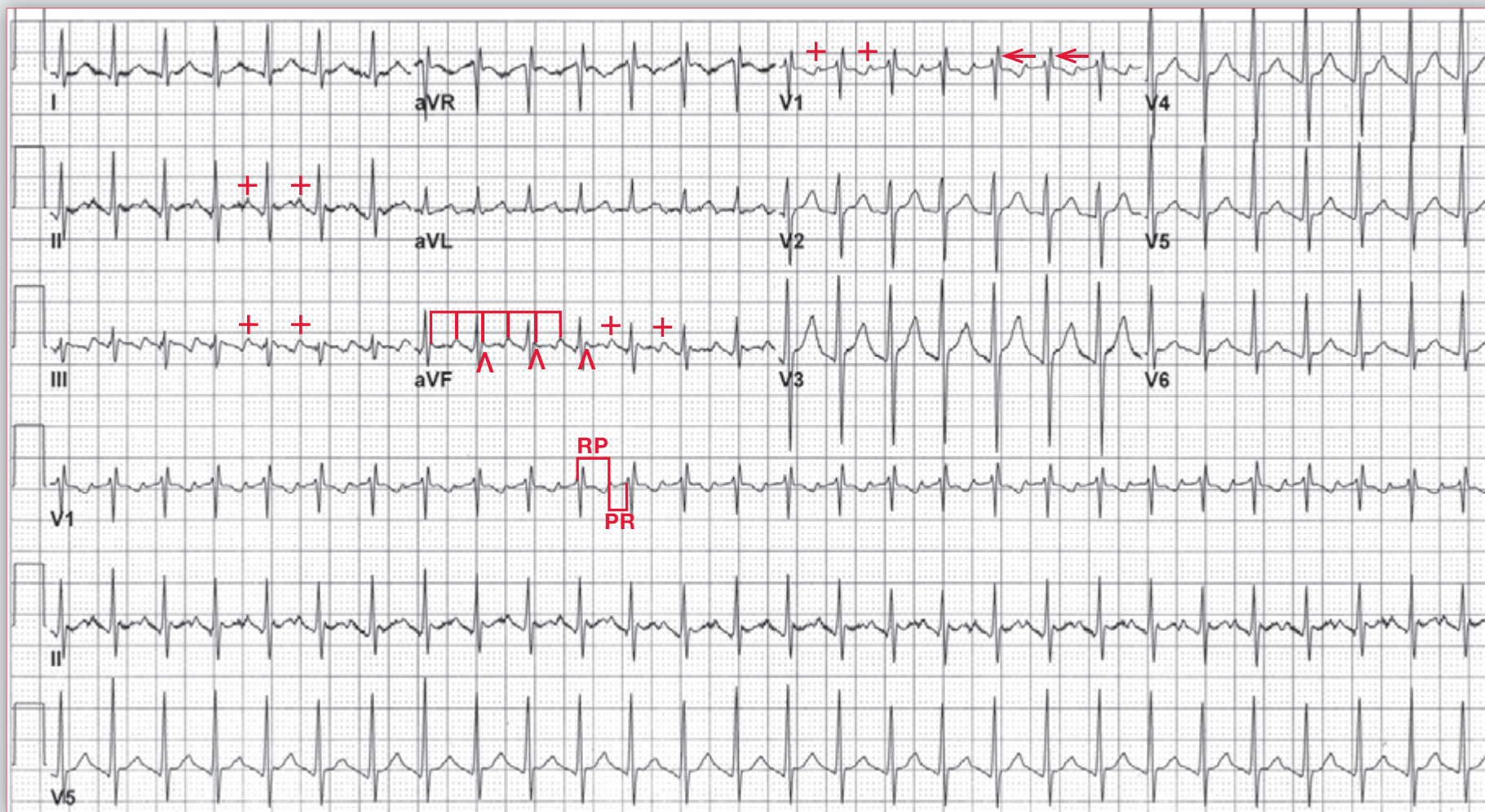


What do the ECGs show?

ECG 49B



Podrid's Real-World ECGs



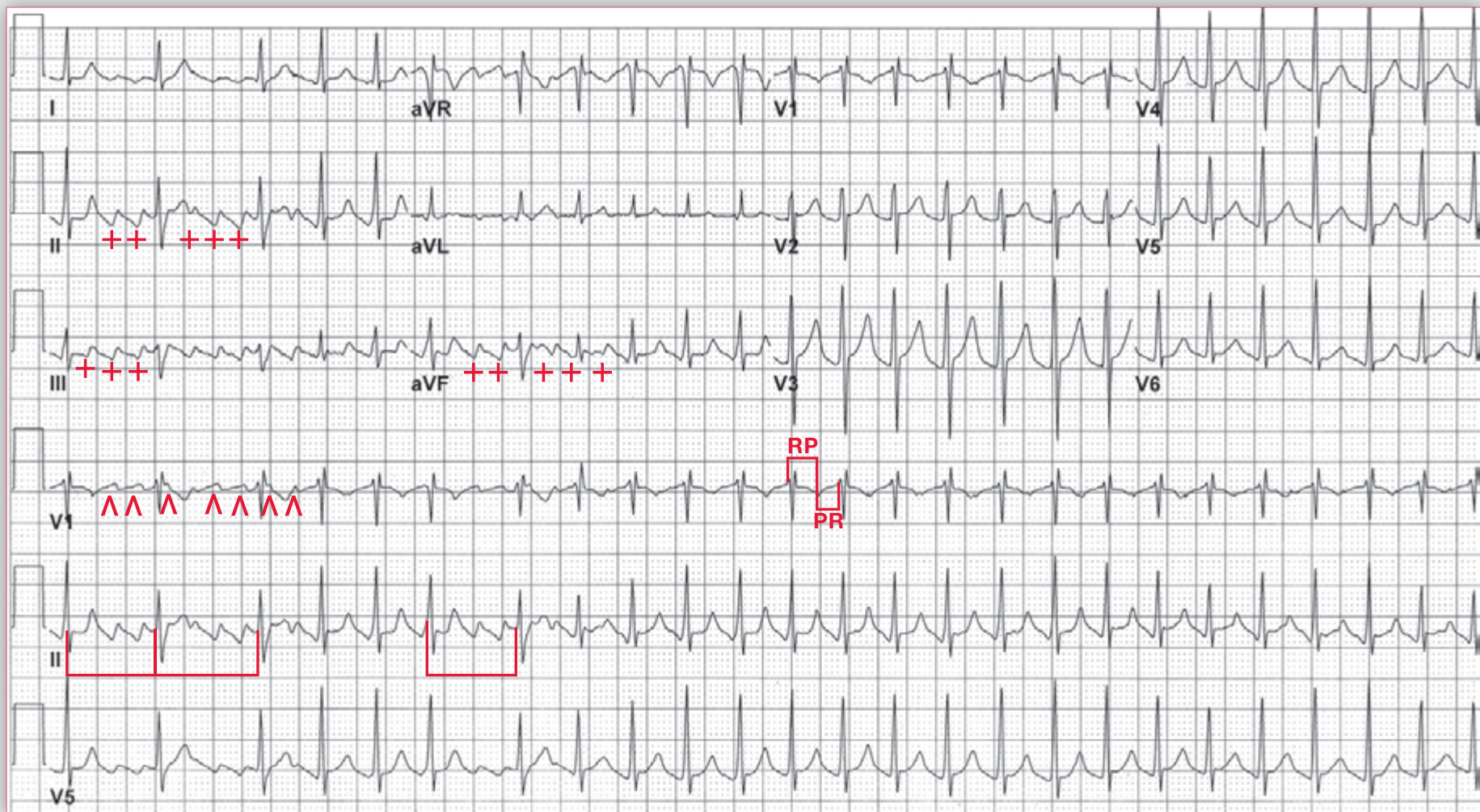
ECG 49A Analysis: Narrow complex supraventricular tachycardia, long-RP tachycardia, atrial flutter with 2:1 AV conduction

ECG 49A shows the rhythm is regular at a rate of 160 bpm. The QRS complex duration is normal (0.08 sec). The QRS complex morphology is normal, although there is an R' in lead V1 (←), representing a minor right ventricular conduction delay. This QRS complex morphology in V1 has been termed a crista pattern, as the last portion of the right ventricle to depolarize is the crista supraventricularis. The axis is about 0° (positive QRS in lead I and biphasic QRS complex in lead aVF). The QT/QTc intervals are normal (270/440 msec).

There are no obvious atrial waveforms seen in any lead. However, there are uniform and regular positive waveforms seen before each QRS complex in leads II, aVF, and V1 (+). In leads III and aVF these waveforms

appear to be negative-positive. This is a long-RP (short PR) tachycardia (RP interval = 0.24 sec, PR interval = 0.12 sec). Etiologies for a long-RP tachycardia include sinus tachycardia, atrial tachycardia, ectopic junctional tachycardia, atypical atrioventricular nodal reentrant tachycardia, atrioventricular reentrant tachycardia or atrial flutter with 2:1 AV block. The P waves seen are negative positive in leads III and aVF, and hence the rhythm is not sinus. With careful inspection of lead aVF, it can be seen that there is a second waveform of the same axis and morphology at the end of the QRS complex (^). The interval between the waveform before and after the QRS complexes in lead aVF is regular at a rate of 320 bpm (□). These findings are consistent with atrial flutter with 2:1 AV conduction.

continues



ECG 49B Analysis: Atrial flutter with variable block

ECG 49B shows the rhythm is regular with an average rate of 150 bpm; however, there are three long RR intervals seen (⏏), accounting for irregularity of the first 7 QRS complexes. The remaining QRS complexes are regular at a rate of 160 bpm, identical to the rate seen in ECG 49A. The QRS complex duration, morphology, and axis are identical to what was seen in ECG 49A. The QT/QTc intervals are the same as ECG 49A.

As a result of the irregularity at the beginning of the ECG, which is due to a greater degree of AV block, there are distinct atrial waveforms seen at a rate of 320 bpm (+). In leads II, III, and aVF these waveforms are negative positive and have a “sawtooth” pattern. In lead V1 the waveforms are positive-negative (^). Hence this is typical atrial flutter with variable block (3:1 and 4:1), ultimately becoming stable with 2:1 AV block. Similar to what was seen in ECG 49A, the rhythm has the appearance of a long-RP (short PR) tachycardia.

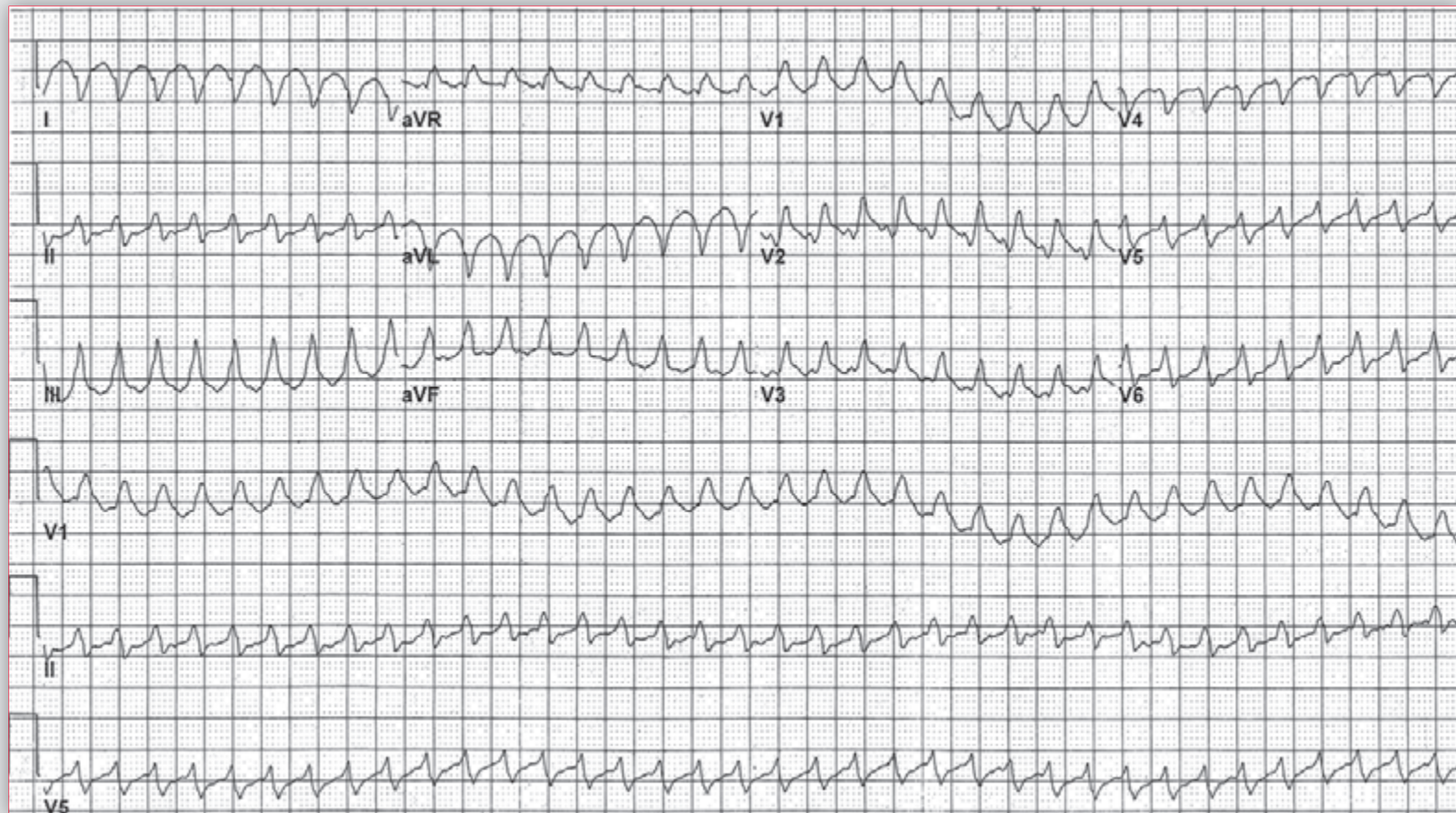
Atrial flutter is often a difficult diagnosis to establish as one of the two flutter waves may be superimposed at the beginning of the QRS complex, resembling a Q wave, or at the end of the QRS complex resembling an S wave or ST-segment depression. In these situations, it may be difficult to distinguish between atrial tachycardia and atrial flutter. This is an important distinction, as atrial flutter generally response to electrocardioversion, which atrial tachycardia (which is most often due to an ectopic focus) is usually not reverted with electrocardioversion. An increase in AV block will expose the atrial waveforms, allowing the etiology of an atrial arrhythmia to be established. This can be achieved with a vagal maneuver (carotid sinus pressure or Valsalva) or the use of an AV nodal blocking agent (adenosine, β -blocker, calcium-channel blocker, or digoxin). ■

Core Case 50

A 54-year-old man, recently discharged after coronary artery bypass surgery, returns to the emergency department with chest pain and palpitations. Physical examination is notable for a faint friction rub. Pulse rate is very rapid, and an ECG is obtained (ECG 50A).

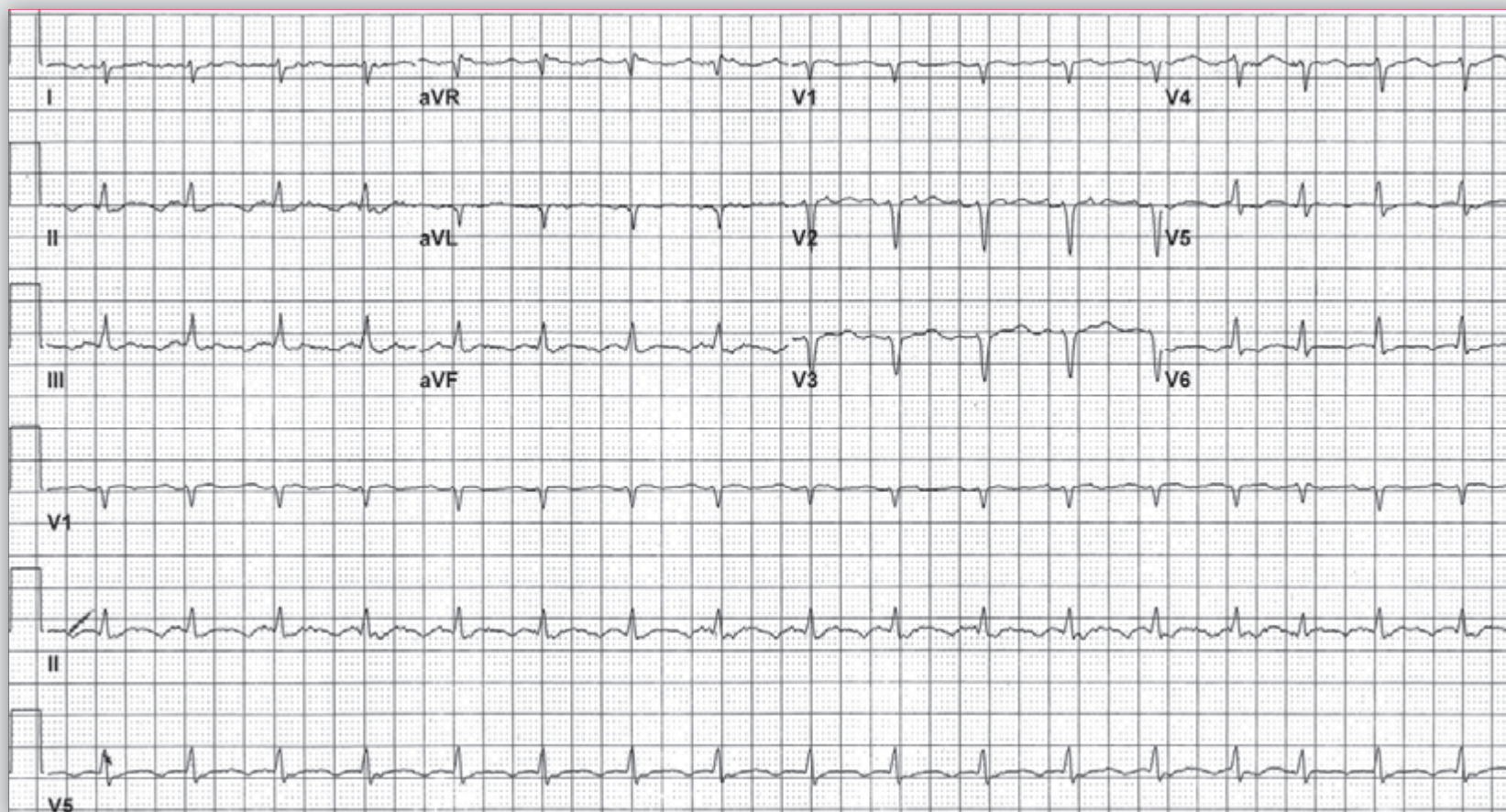
As a result the patient is transferred to the coronary care unit with concerns for an acute coronary syndrome

ECG 50A



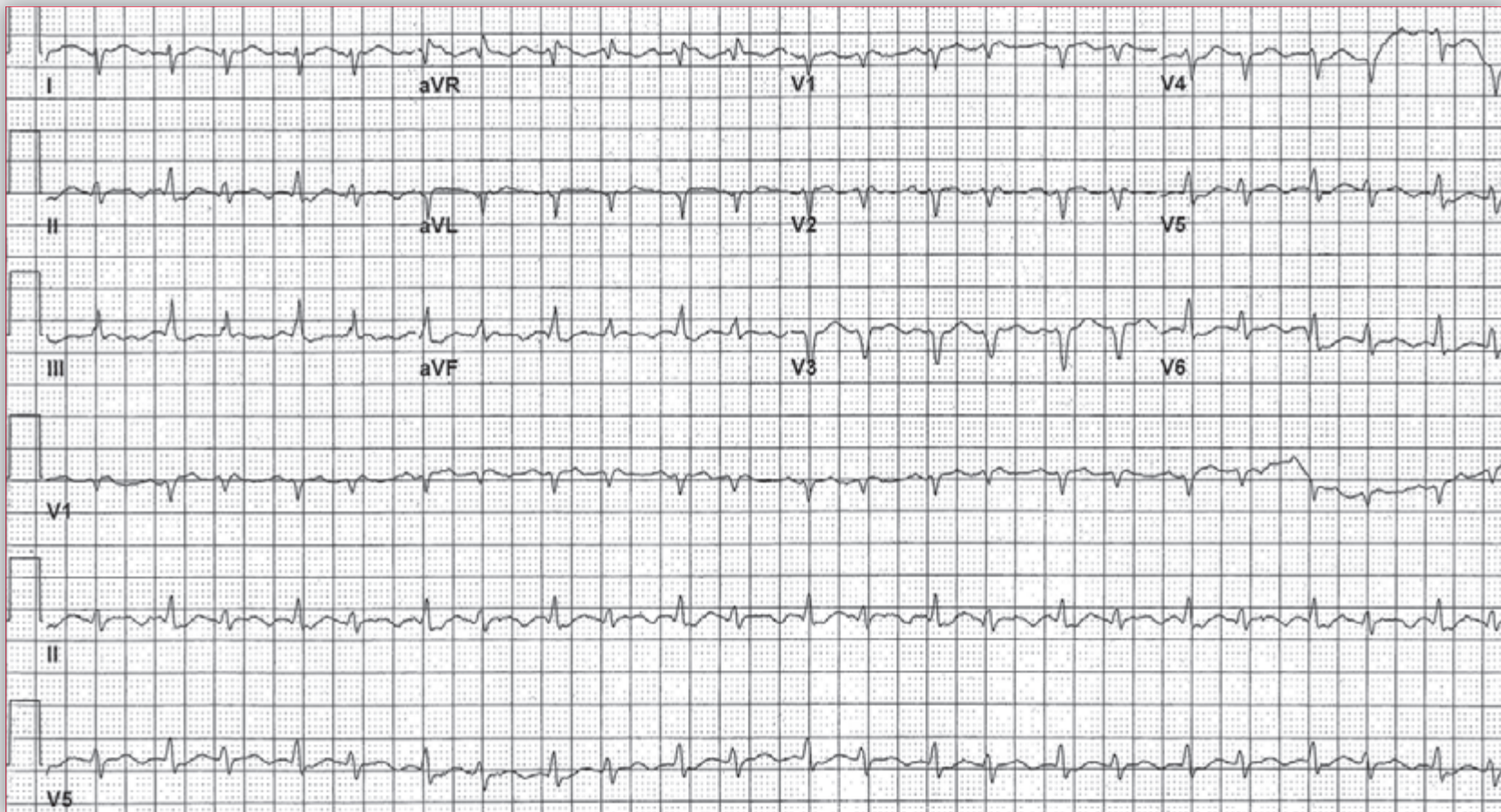
associated with ventricular tachycardia. Shortly after being admitted to the coronary care unit, another ECG is obtained (ECG 50B). Cardiac enzymes are negative, and an echocardiogram demonstrates intact LV function, unchanged from prior to surgery. A small pericardial effusion is seen. However, while the echocardiogram is being recorded, there is a change in his rhythm. Another ECG is obtained (ECG 50C).

ECG 50B



Core Case 50

ECG 50C



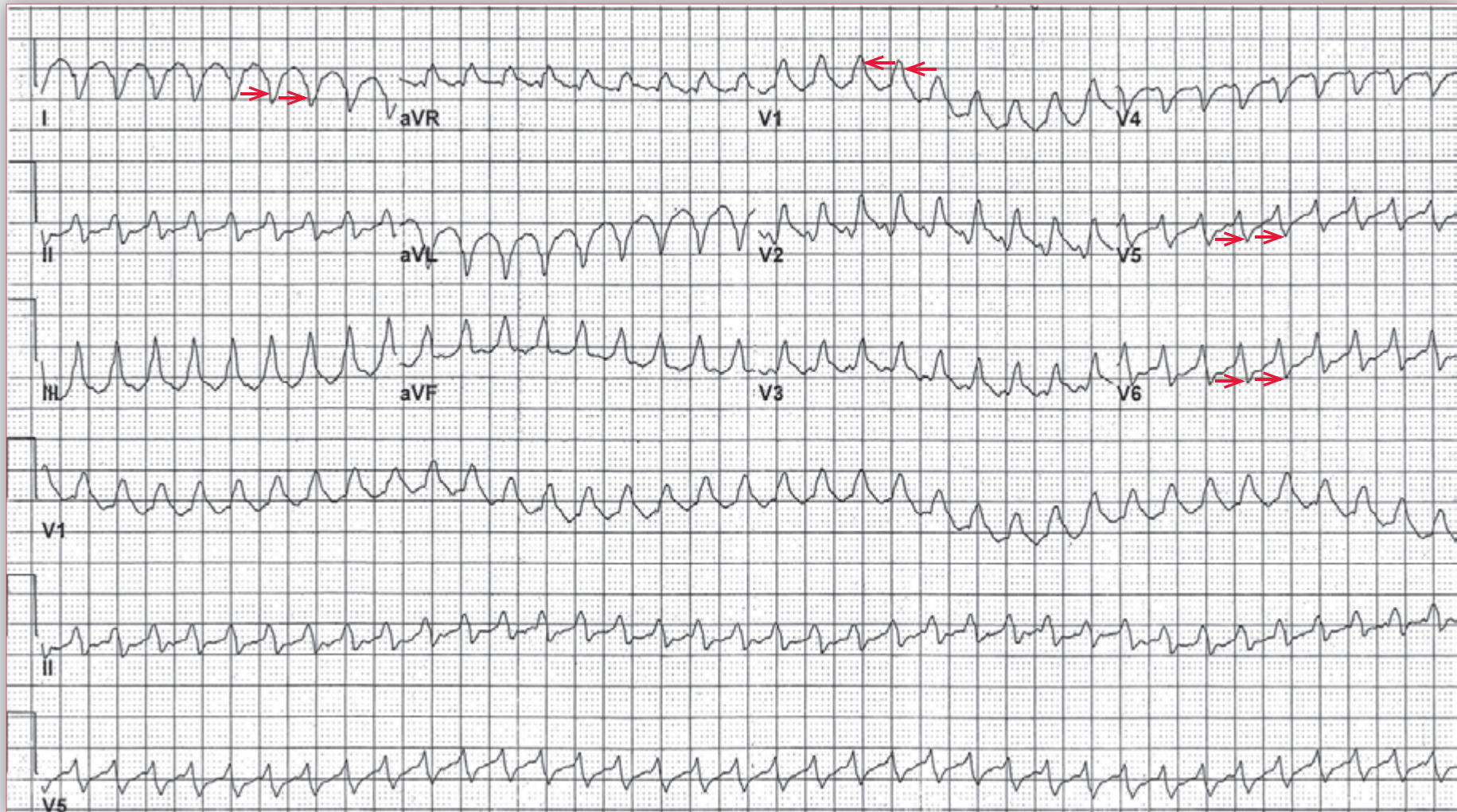
What possible arrhythmias are suggested by ECG 50A?

What does ECG 50B show?

What is the cause for the changes in the ECGs?

What is the likely diagnosis, and what therapy should be started?

Podrid's Real-World ECGs



ECG 50A Analysis: Regular wide complex tachycardia (atrial flutter with 1:1 conduction), right bundle branch block (rate-related), and right axis deviation (left posterior fascicular block), low voltage

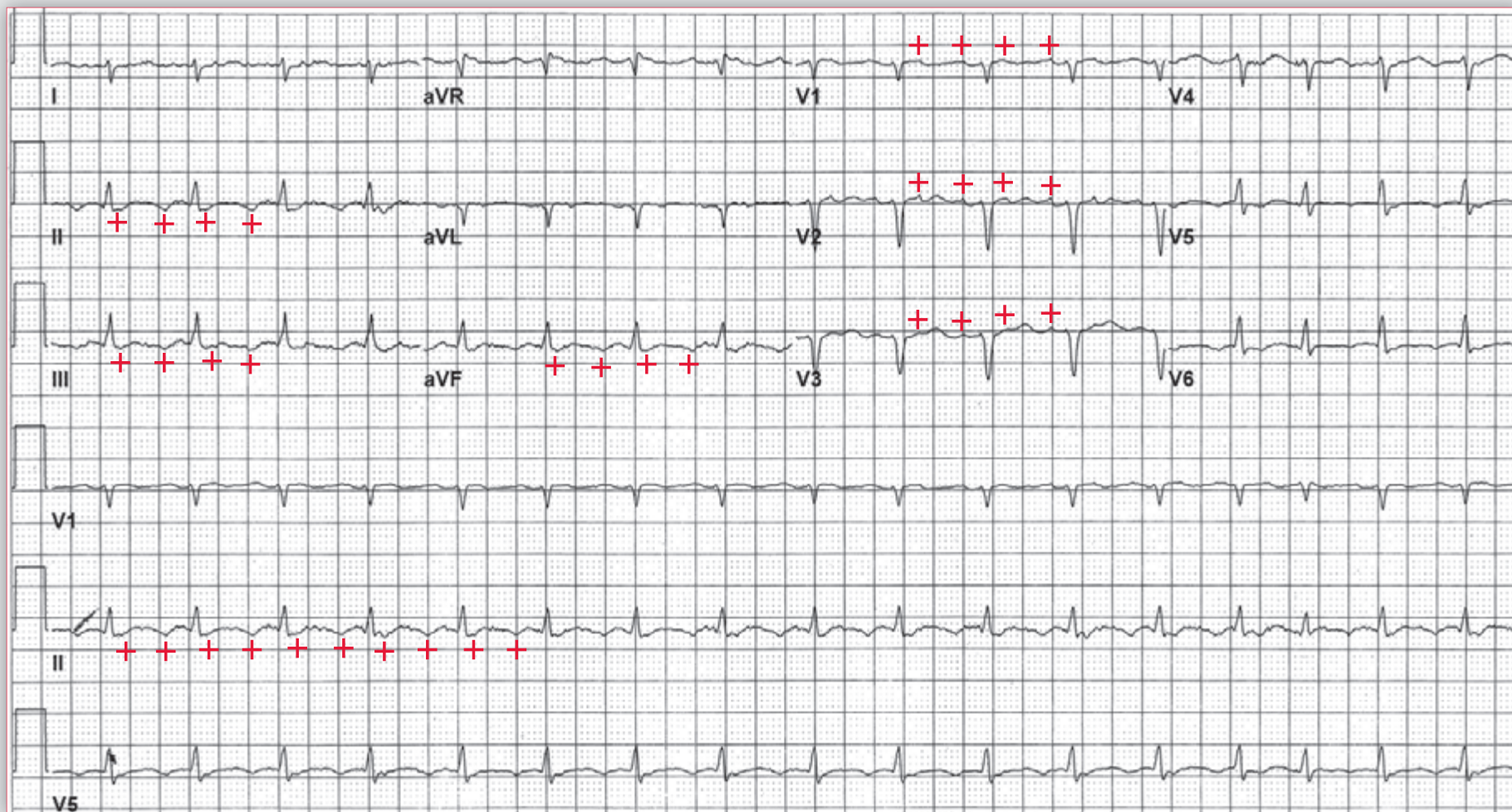
ECG 50A shows a regular rhythm at a rate of 260 bpm. There are no distinct P waves noted. The QRS complex duration is increased (0.12 sec) and there is a right bundle branch block morphology with a broad R wave in V1 (←) and broad S waves in leads I and V5–V6 (→). In addition there is a rightward axis between $+90^\circ$ and $+180^\circ$ (negative QRS complex in leads I and positive QRS complex in lead aVF). This is suggestive of a left posterior fascicular block, although an old lateral

wall myocardial infarction is suggested by the possible QS complex morphology in leads I and aVL. This rhythm may be either atrial flutter with 1:1 AV conduction or ventricular tachycardia, which at this rate is often referred to as ventricular flutter.

In addition, there is low QRS voltage (< 5 mm or little boxes in each limb lead and < 10 mm in each precordial lead).

continues

Podrid's Real-World ECGs



ECG 50B Analysis: Narrow complex supraventricular tachycardia, atrial flutter
2:1 block, left posterior fascicular tachycardia, low voltage

ECG 50B is from the same patient as ECG 50A. There is a regular rhythm at a rate of 130 bpm. The QRS complex duration is normal (0.08 sec). The axis is rightward between $+90^\circ$ and $+180^\circ$ (negative QRS complex in lead I and positive QRS complex in lead aVF). In the absence of any reason for the rightward axis (*ie*, lateral wall MI, dextrocardia, right ventricular hypertrophy, Wolff-Parkinson-White, or R-L arm lead switch) this is a left posterior fascicular block. There is low QRS voltage (< 5 mm or little boxes in each limb lead and < 10 mm in each precordial lead). The QT/QTc intervals are prolonged (320/470 msec).

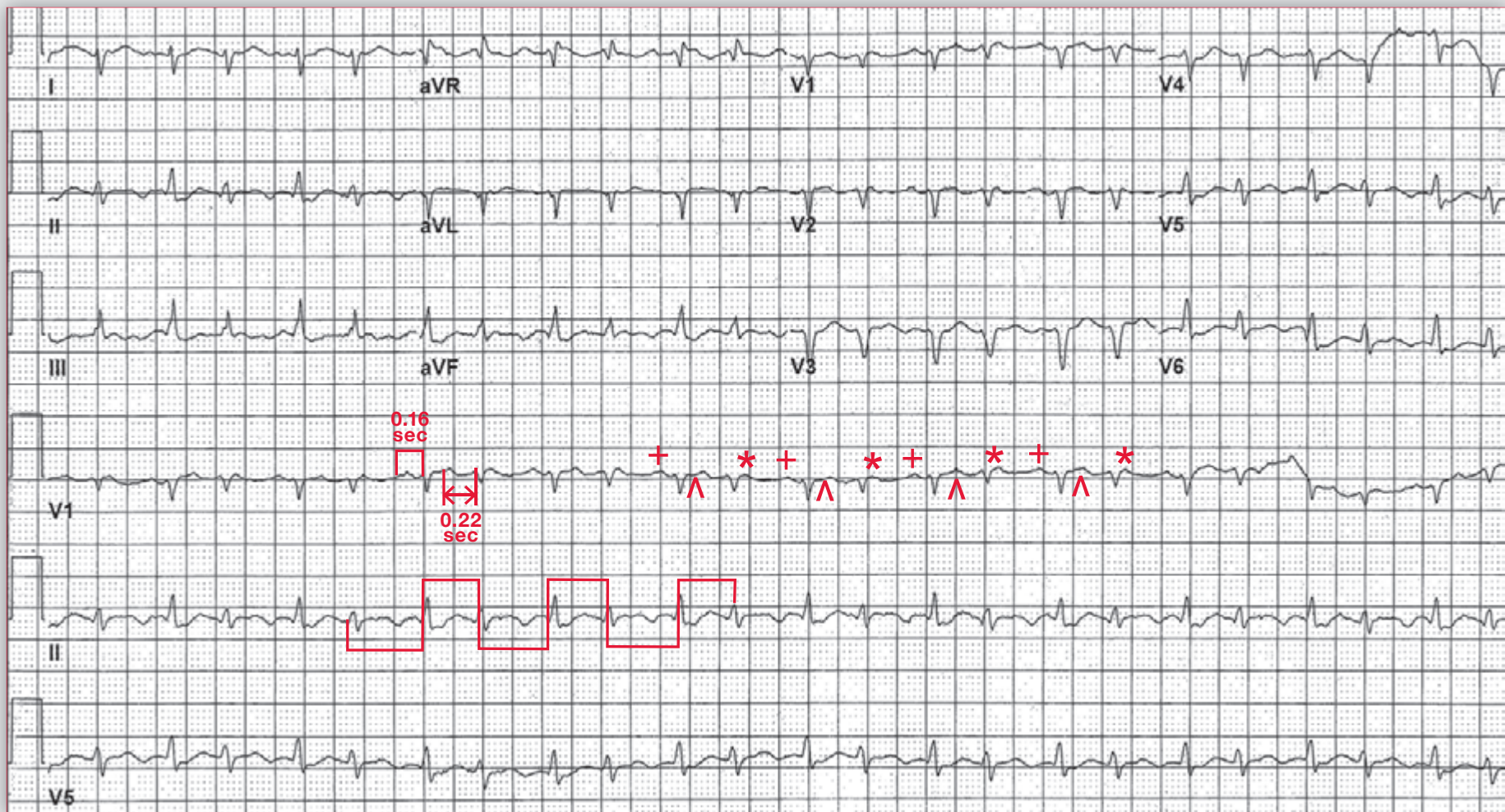
Evidence of atrial activity can be seen in leads II, III, aVF, and V1–V3 (+). The atrial waveforms are undulating without any isoelectric baseline between them. The atrial rate is 260 bpm and hence this is atrial flutter with 2:1 AV conduction. The atrial rate is identical to the ventricular rate seen in ECG 50A, and hence the rhythm is indeed

atrial flutter with 1:1 conduction. An important concept is “same rate same rhythm.” A right bundle branch block morphology is not seen on ECG 50B, indicating that ECG 50A had a rate-related right bundle branch block.

Atrial flutter with 1:1 conduction is not common and generally reflects the presence of high sympathetic tone or elevated catecholamine levels that enhance AV nodal conduction. Situations in which this occurs include hyperthyroidism, post exercise, severe infection/sepsis, heart failure, and pulmonary embolism. When the QRS complex is narrow, this diagnosis is easier to establish, as any regular narrow complex tachycardia at a rate > 260 is atrial flutter with 1:1 conduction. When the QRS complex is wide, the arrhythmia may be either supraventricular with a rate-related or preexistent bundle or it may be ventricular tachycardia. It may be difficult to distinguish between these two arrhythmias.

continues

Podrid's Real-World ECGs



ECG 50C Analysis: Atrial flutter with 3:2 Wenckebach, left posterior fascicular block, low voltage

ECG 50C is from the same patient as ECGs 50A and 50B. The rhythm is regularly irregular with an average rate of 144 bpm. There is group beating seen, with a repeating pattern of long (⏏) and short (⏏) intervals. Therefore the rhythm is regularly irregular. The QRS complex duration, morphology, and axis are the same as seen in ECG 50B. The QT/QTc intervals are the same.

There are distinct atrial waveforms seen, especially in lead V1. There is an atrial waveform seen before each of the QRS complexes after the long RR interval (+). There is also an atrial waveform seen before the next QRS complex (^), which is after the short interval. The intervals of these two atrial waves is regular with an atrial rate of 260 bpm, similar to the atrial rate seen in leads 50A and 50B. Using this interval, it can be seen that there is an atrial waveform at the end of the QRS after the short RR interval and before the pause (*). It can be seen that the morphology of this QRS complex is different as the terminal portion is rounded, due to the superimposed atrial waveform. The underlying rhythm is atrial flutter at a rate of 260 bpm. It can be seen that after the long RR interval, the flutter wave (+) to QRS complex interval is 0.16 second. The next flutter wave (^) to QRS complex interval is

0.22 second, while the third flutter wave (*) is nonconducted. Hence this is a pattern of 3:2 Wenckebach. Mobitz type I second-degree AV block, or Wenckebach, is a result of decremental conduction through the AV node. In this situation there is progressive lengthening of the PR interval with one nonconducted P wave after which the PR interval shortens back to its baseline. Wenckebach can be seen with sinus rhythm, atrial tachycardia, or atrial flutter.

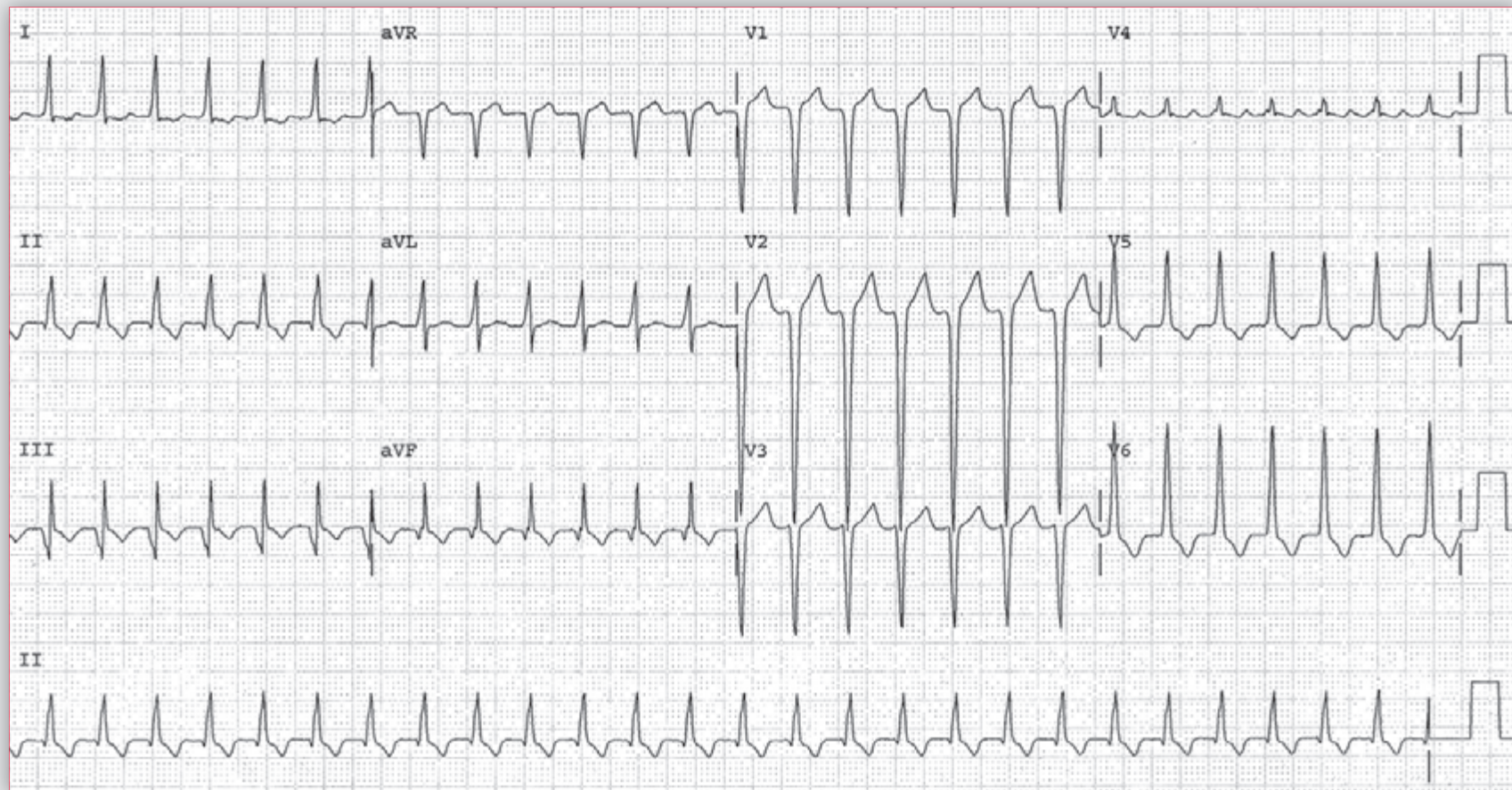
The clinical presentation of this patient suggests a post-pericardiotomy syndrome, resulting from recent bypass surgery and pericardial trauma with the release of cardiac antigens. This produces an immunologic reaction that produces a pericarditis (indicated by the chest pain, friction rub, and small pericardial effusion). Also seen with this condition are a pleuritis, arthralgias, arthritis, and fever. This syndrome is the same as is seen after an acute myocardial infarction, known as Dressler's syndrome. The pericarditis is a stimulus for atrial arrhythmias, including atrial flutter.

Treatment for the pericarditis and the associated pain included colchicine, NSAIDs, and on rare occasions steroids. ■

Core Case 51

A 64-year-old man is admitted with palpitations. He states that he was told of a previous heart attack, but is not experiencing any symptoms except for the palpitations. Shortly after arrival, the arrhythmia terminates. ECGs before and after termination are shown.

ECG 51A

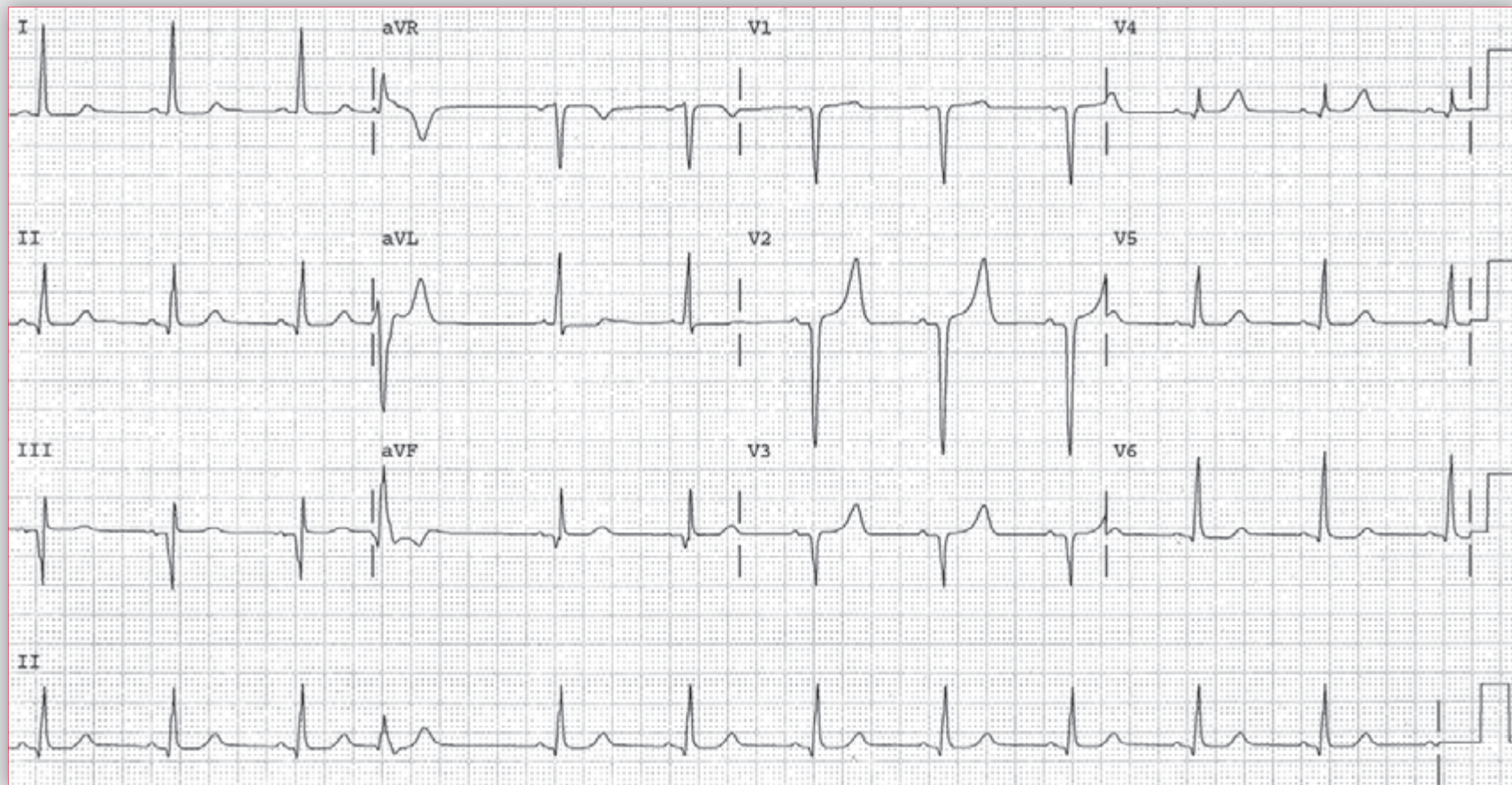


What do the ECGs show?

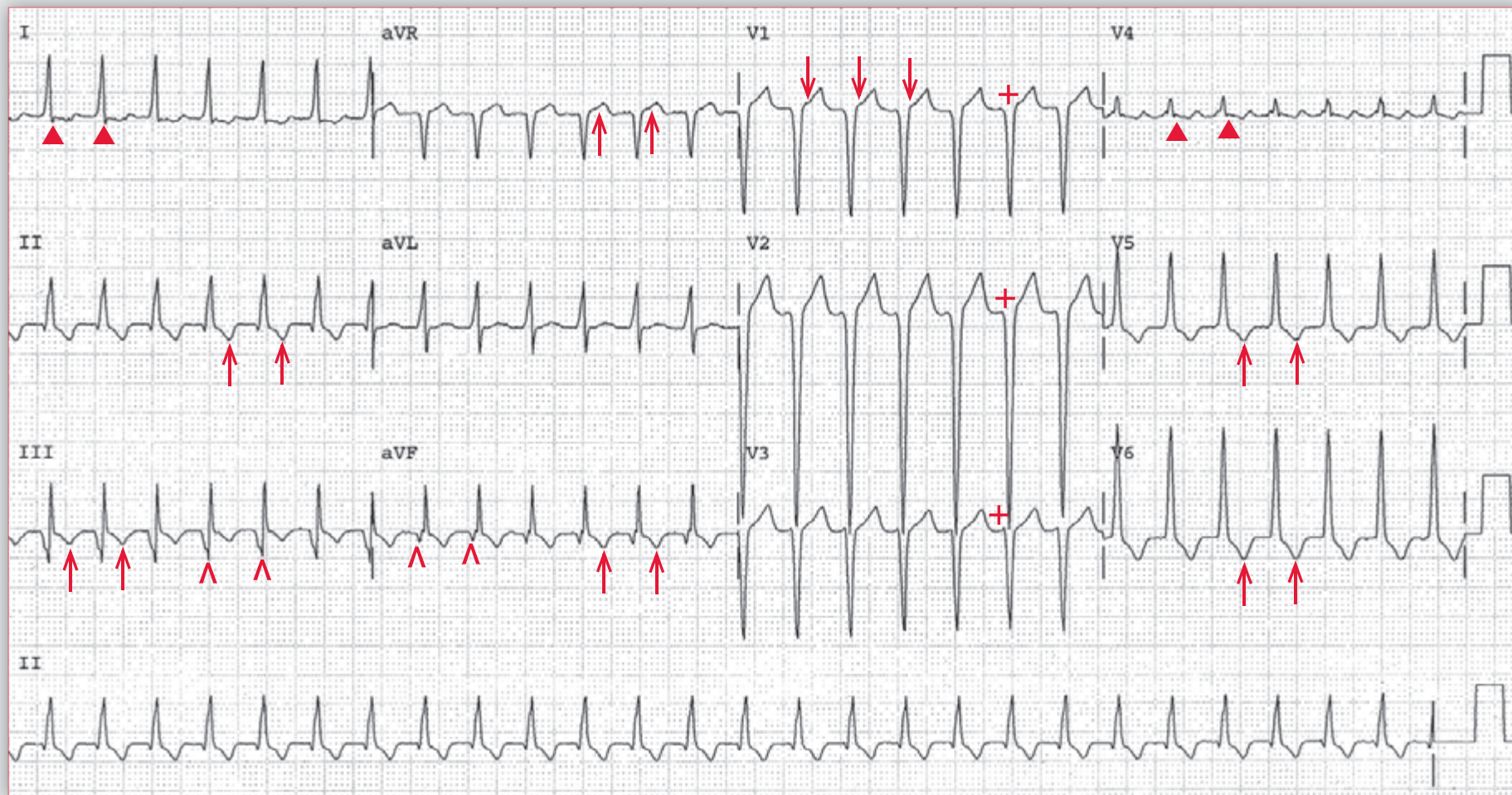
What are the clues to the underlying diagnosis?

What therapy would be appropriate?

ECG 51B



Podrid's Real-World ECGs



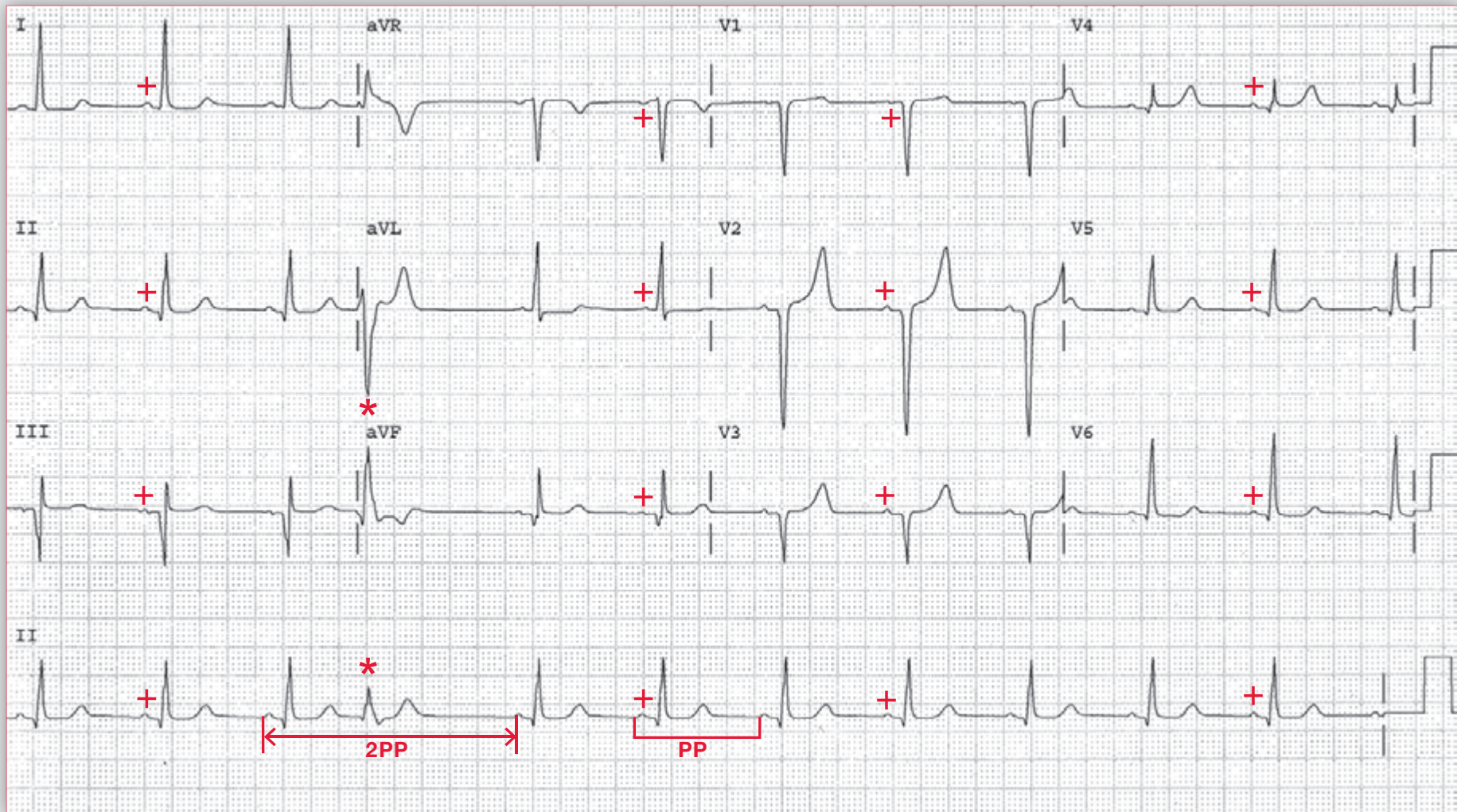
ECG 51A Analysis: No-RP tachycardia, atrioventricular nodal reentrant tachycardia, possible old inferior myocardial infarction, old anteroapical myocardial infarction

ECG 51A shows a regular rhythm at a rate of 160 bpm. The QRS complex duration is normal (0.10 sec) and the axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). There are Q waves in leads III and aVF (\wedge), consistent with an old inferior wall myocardial infarction. In addition there is a QS morphology in leads V1–V3 ($+$), consistent with an old anteroseptal myocardial infarction. The QT/QTc intervals are slightly prolonged (280/460 msec). There are nonspecific T-wave inversion in leads II, III, aVF, and V5–V6 (\uparrow). The positive T wave in lead aVR is actually T-wave inversion.

There are no obvious P waves seen before or after any of the QRS complexes. In lead V1, there is a subtle notching at the very end of the QRS complex (\downarrow), which is suggestive of a superimposed P wave. In addition, there are subtle waveforms after the QRS complex in leads I (small narrow S wave) and V4 (\blacktriangle). However, these are not distinct atrial waveforms. This could be termed a no-RP tachycardia. The most common etiology for this type of supraventricular tachycardia is an atrioventricular nodal reentrant tachycardia. It is very uncommon for an atrial tachycardia or an atrioventricular reentrant tachycardia to present in this fashion.

continues

Podrid's Real-World ECGs



ECG 51B Analysis: Normal sinus rhythm, old inferior and anteroseptal myocardial infarction

ECG 51B is from the same patient as ECG 51A. There is a regular rhythm at a rate of 66 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.16 sec). The P waves are positive in leads I, II, aVF, and V4–V6. Hence this is a normal sinus rhythm. The QRS complex duration, axis, and morphology is the same as seen in ECG 51A; there is an old inferior and anteroseptal myocardial infarction. The notching seen at the end of the QRS complex in lead V1, the small S wave at the end of the QRS complex in lead I and the positive deflection after the QRS complex in lead V4 are not present, suggesting that these were P waves. The QT/QTc intervals are the same as ECG 51A.

The fourth QRS complex is premature (*). It is wider and has a different morphology compared to the sinus QRS complexes. There is no P wave before this complex. Hence this is a premature ventricular

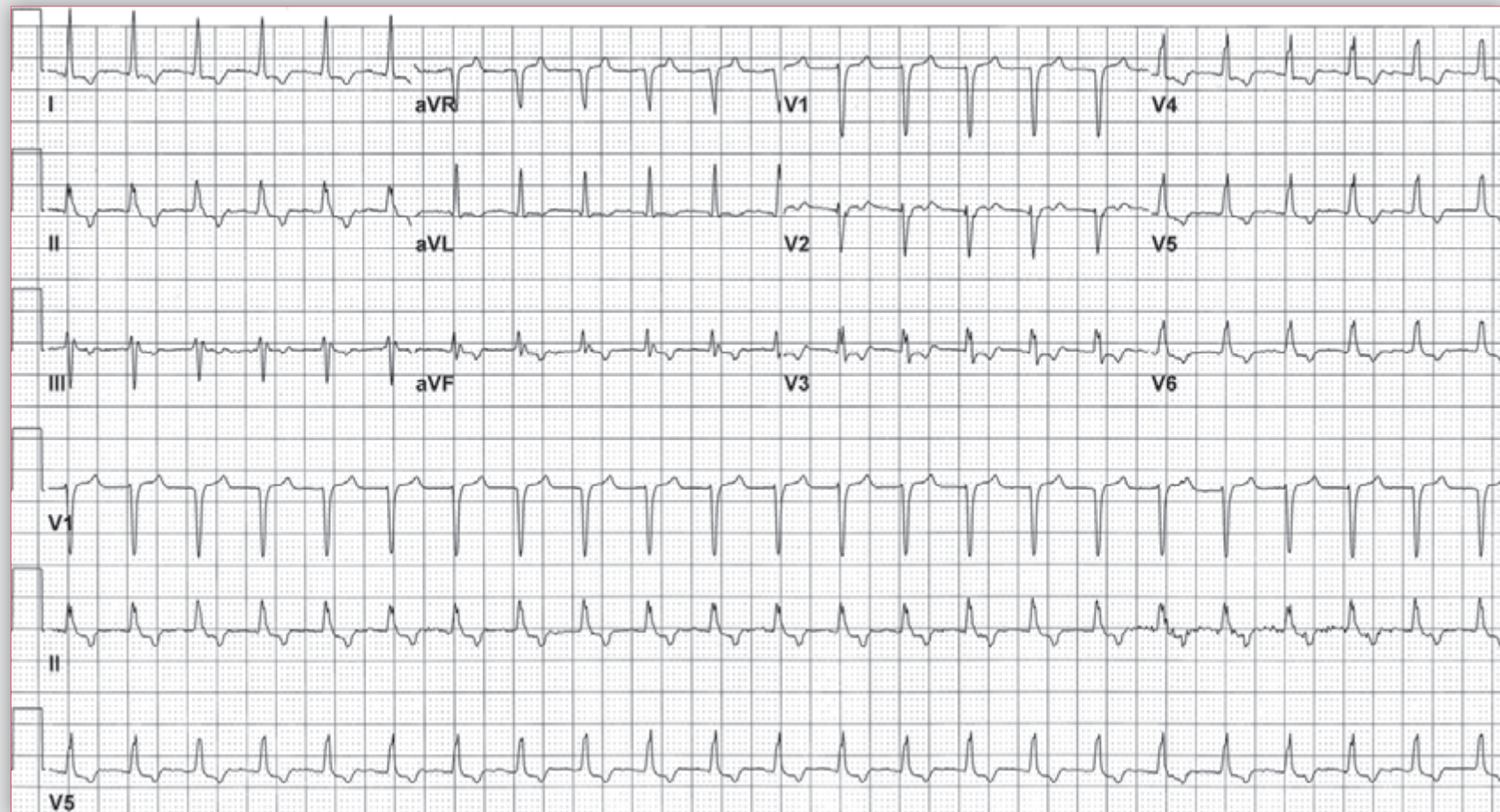
complex. There is an associated full compensatory pause, *ie*, the PP interval surrounding the premature ventricular complex is twice the underlying sinus rhythm (\leftrightarrow).

As indicated the arrhythmia in ECG 51A is most consistent with a typical atrioventricular nodal reentrant tachycardia with antegrade activation of the ventricles via a slow pathway and simultaneous retrograde activation of the atria via a fast pathway (hence called slow-fast). Acute treatment for this arrhythmia involves altering the electrophysiologic characteristics of either pathway, *ie*, a vagal maneuver (such as Valsalva or carotid sinus pressure) or an AV nodal blocking agent such as adenosine, β -blocker, calcium-channel blocker (diltiazem or verapamil), or digoxin. Long-term therapy involves the use of an AV nodal blocking agent or the use of AV nodal ablation with radiofrequency waves. ■

Core Case 52

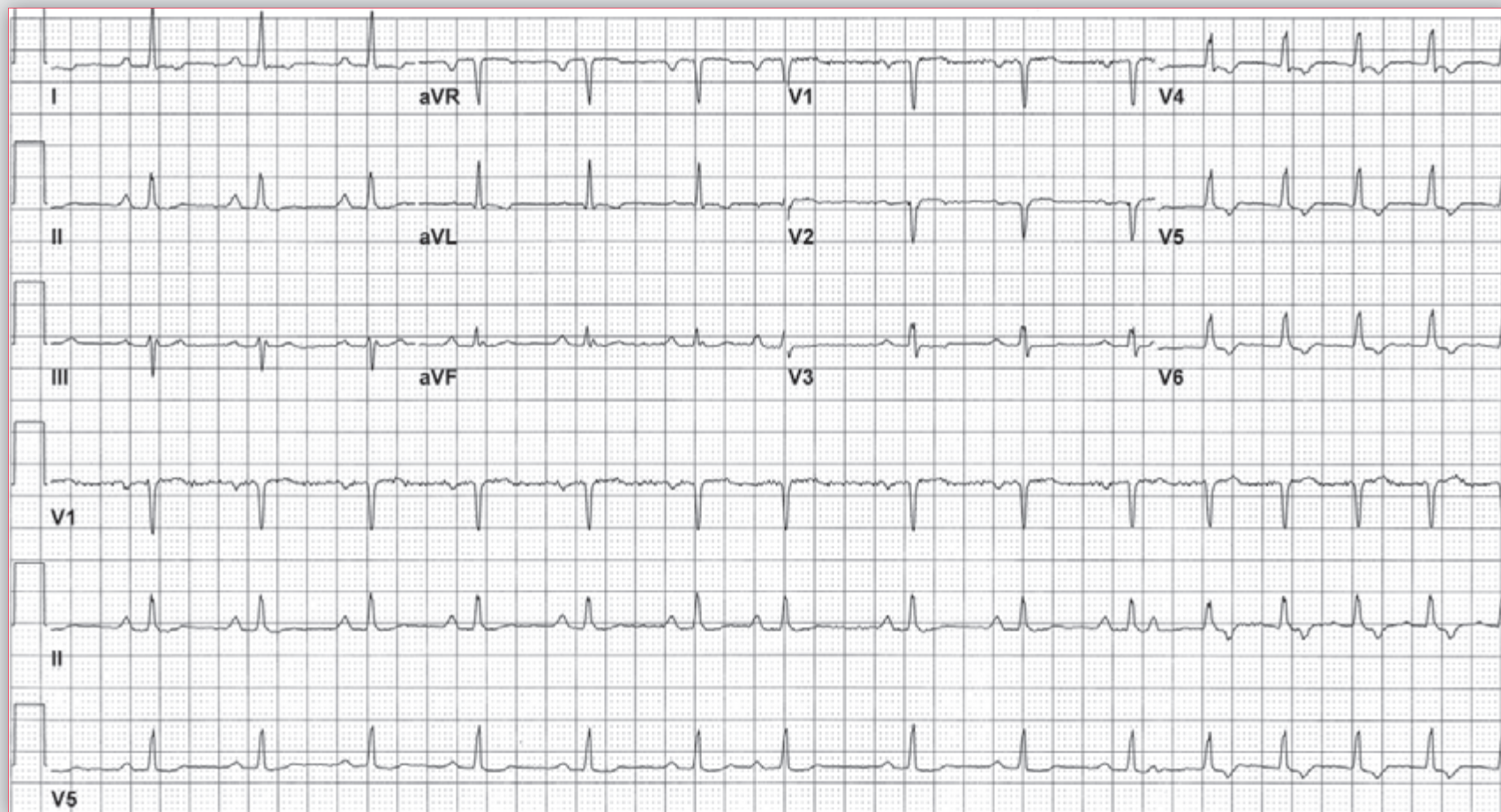
A 68-year-old man has had palpitations since the age of 25. Initially the episodes were infrequent, about once per year. They were usually short-lived, lasting about 30 to 60 minutes. However, the frequency of the episodes has recently increased, and just prior to presentation, he had an episode that began several hours before being seen. His physical examination is unremarkable. An ECG was obtained

ECG 52A



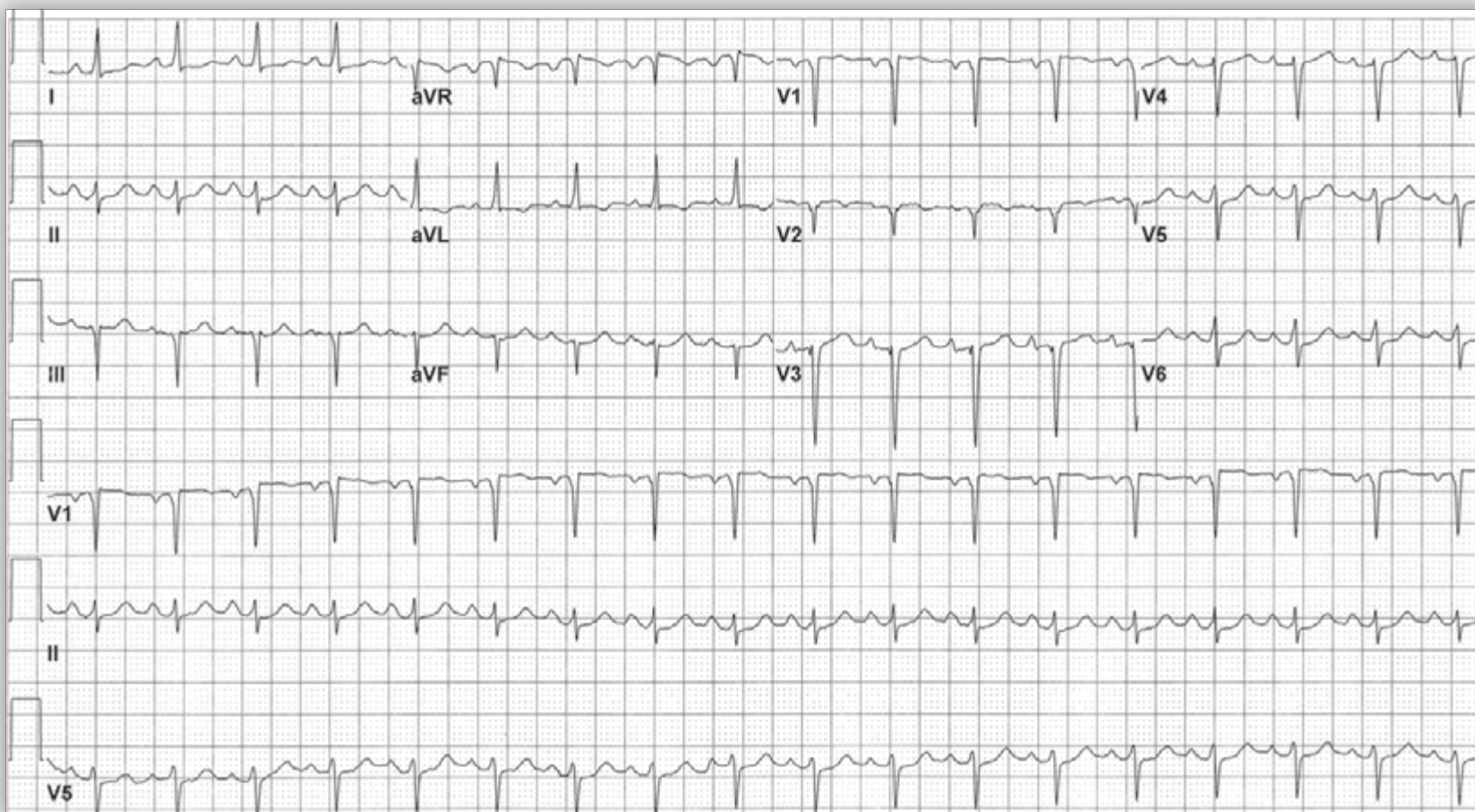
(ECG 52A). Shortly after the ECG was obtained, therapy was given and the tachycardia terminated, at which time the symptoms abated. However, while recording another ECG, the patient again noted the onset of his symptoms (ECG 52B). He was again treated, and after treatment, another ECG is obtained (ECG 52C). The ECGs provide enough information to make a diagnosis.

ECG 52B



Core Case 52

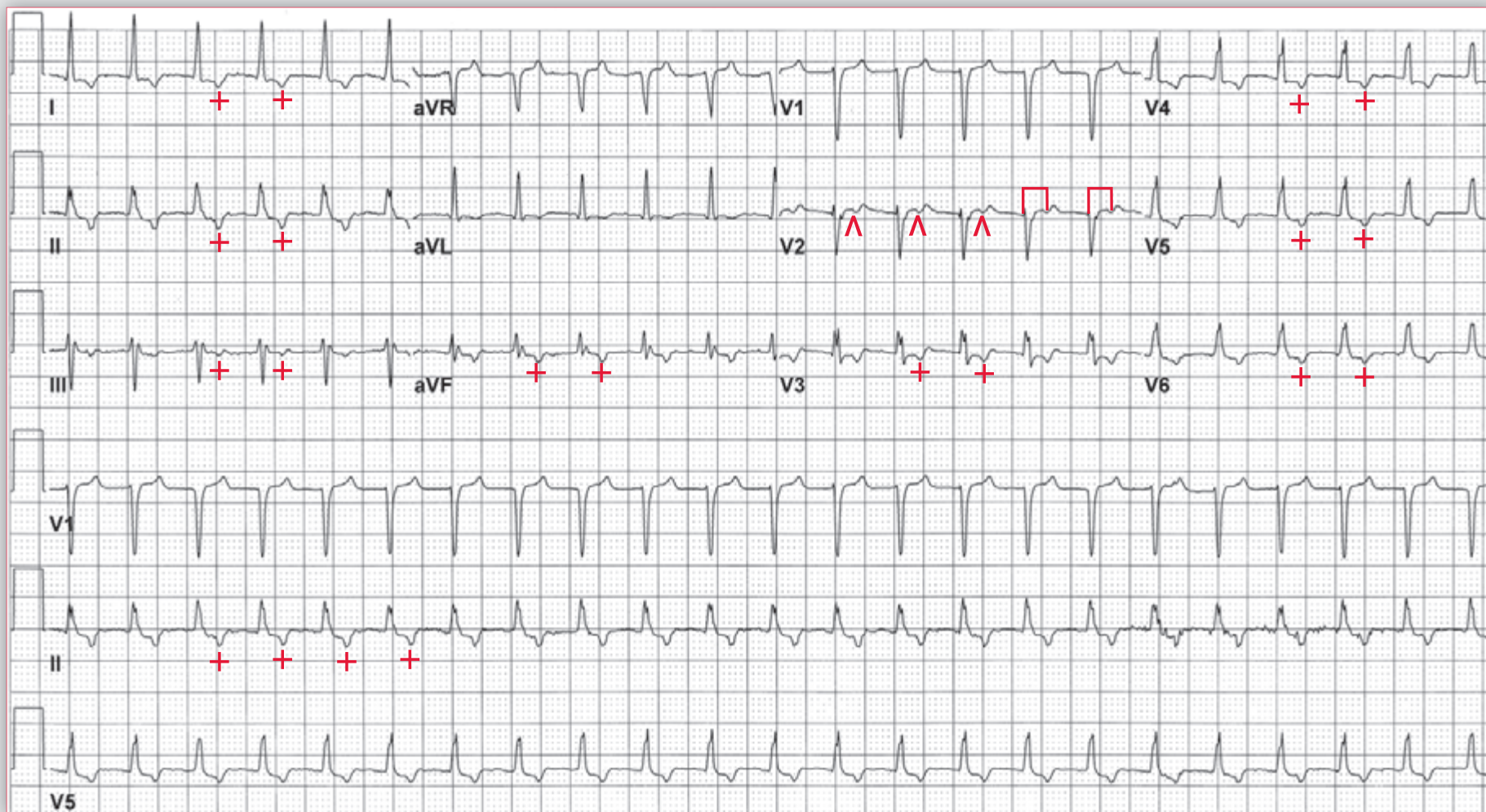
ECG 52C



What do the ECGs show?

What is the likely diagnosis?

Podrid's Real-World ECGs



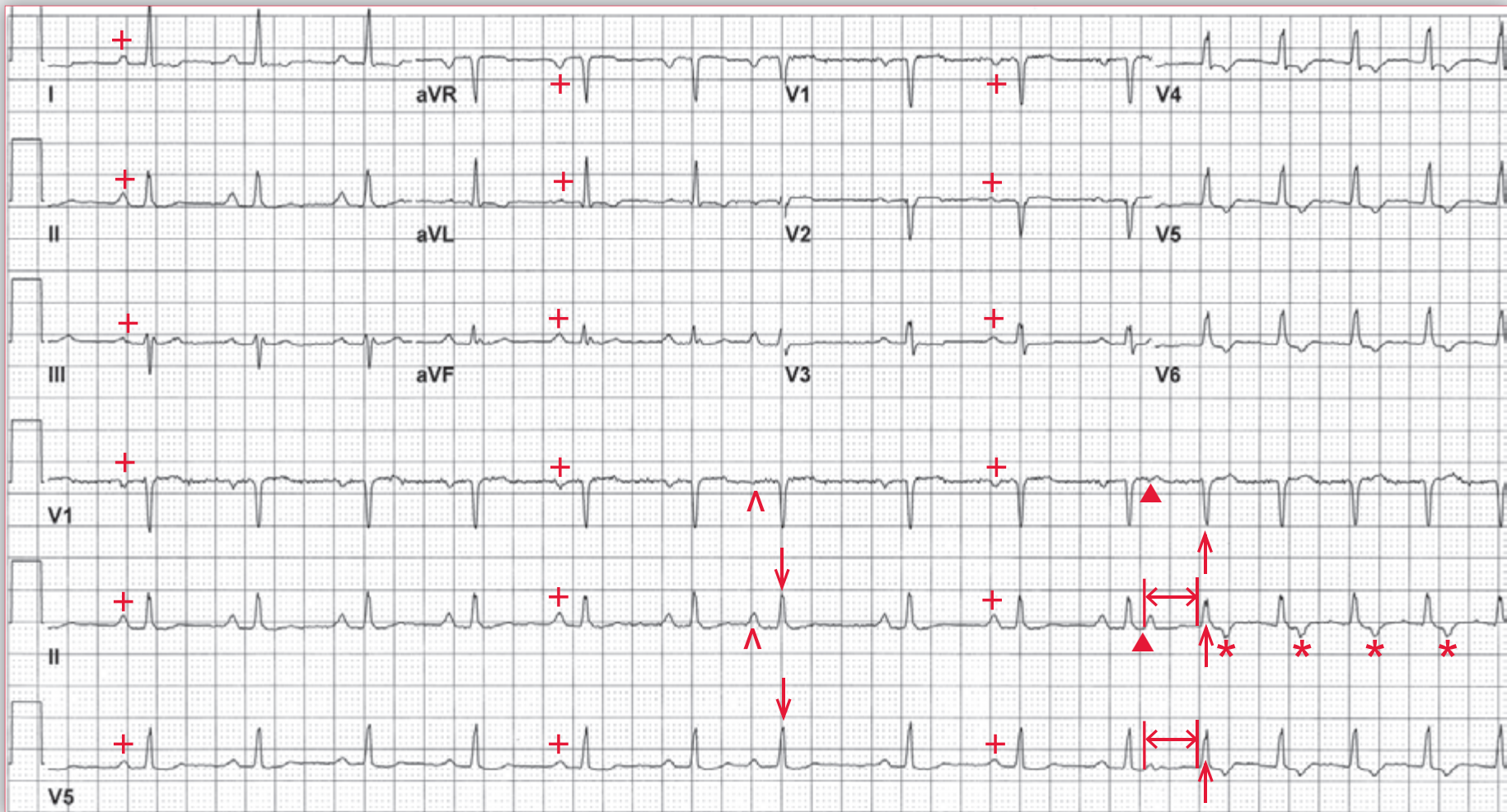
ECG 52A Analysis: Narrow complex supraventricular tachycardia (short-RP tachycardia),
typical atrioventricular nodal reentrant tachycardia (slow-slow)

ECG 52A shows a regular rhythm at a rate of 140 bpm. The QRS complex duration is normal (0.08 sec) and the axis normal between 0° and $+90^\circ$ (positive QRS complex in lead I and aVF). The QT/QTc intervals are normal (280/430 msec). There are no obvious P waves before or after any QRS complexes. However, there is notching of the ST segments noted in lead V2 (^), which appears to be a negative waveform; this is probably a negative P wave. The RP interval is constant (0.16 sec) (□). Based on this interval, it can be seen that the negative deflections after the QRS complexes in leads I, II, aVF, and V3–V6 (+) are not T waves but are the same waveform with the same RP interval. Hence these are negative P waves. Therefore, this is a short-RP

tachycardia (RP = 0.16 sec, PR = 0.28 sec). The etiologies for a short-RP tachycardia include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, an atrioventricular reentrant tachycardia, and an unusual type of a common atrioventricular nodal reentrant tachycardia (slow-slow). In this type of AVNRT, retrograde conduction via the fast pathway is relatively slow, as a result of a drug effect or disease of this tract. Since the P waves are negative in leads II, III, aVF, this is not a sinus tachycardia. In addition, there is no other atrial waveform noted, making atrial flutter unlikely. However, the etiology cannot be definitely established, and it may be an atrial tachycardia or an arrhythmia related to the AV node.

continues

Podrid's Real-World ECGs



ECG 52B Analysis: Normal sinus rhythm, premature atrial complexes, narrow complex supraventricular tachycardia (short PR tachycardia), atrioventricular nodal reentrant tachycardia (slow-slow variant)

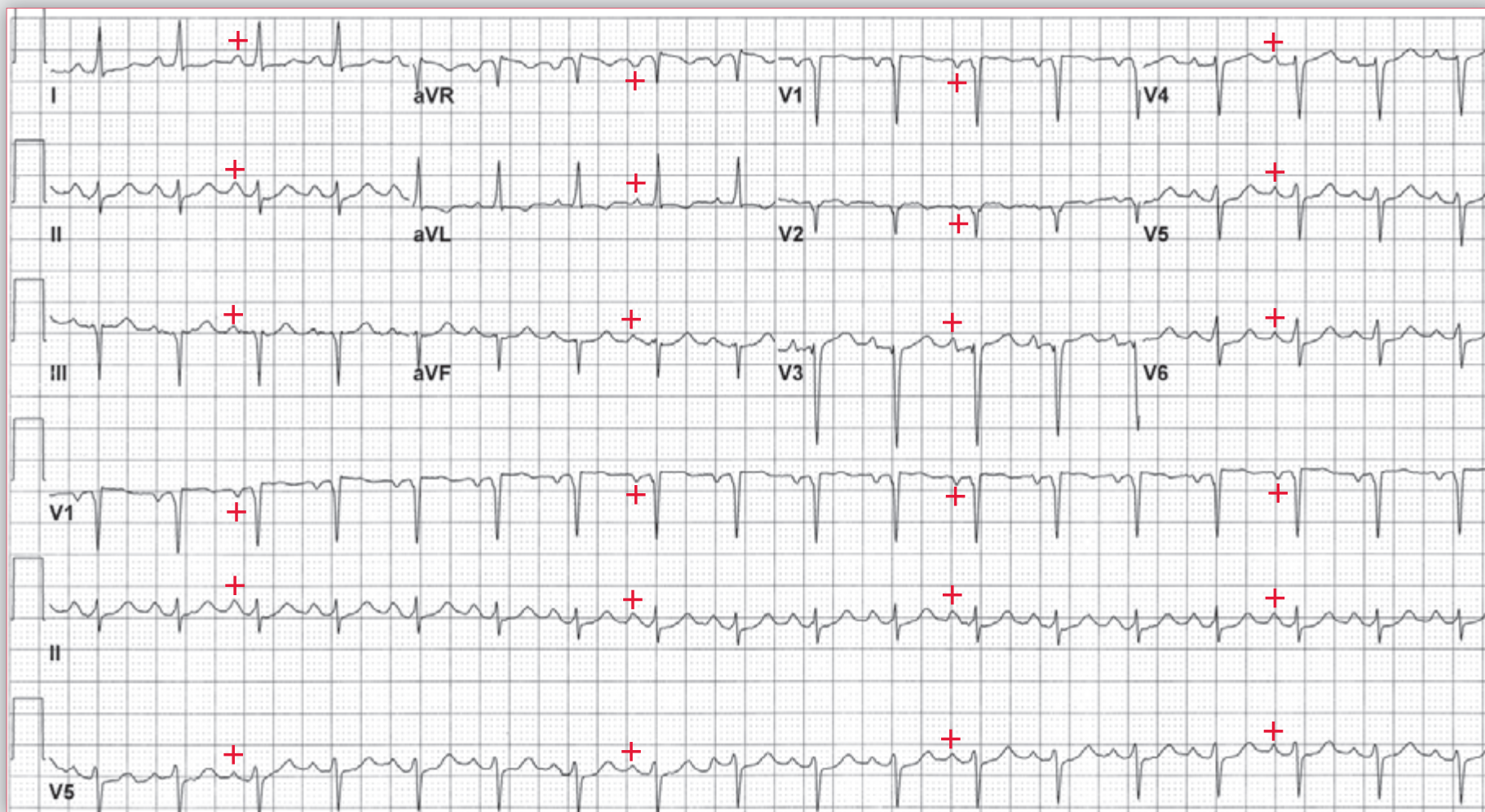
ECG 52B is from the same patient as ECG 52A. The QRS complex duration, morphology, and axis are the same as in ECG 52A. The QT/QTc intervals are normal (340/410 msec). There is basically a regular rhythm at a rate of 88 bpm. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P waves are positive in leads I, II, and aVF. Although no P waves can be seen before the QRS complexes in leads V4–V6, this is probably a normal sinus rhythm. By comparison with the QRS complexes and ST segments seen in ECG 52A, it can be seen that the negative waveforms seen after the QRS complexes are indeed P waves, as they are not present in the sinus complexes.

The seventh QRS complex is premature (↓) and is preceded by a P wave (^) that has a morphology and PR interval (0.22 sec) that are different compared with the sinus complexes. Hence this is a premature atrial complex. The eleventh QRS complex is also premature (↑), and there is a premature P wave (▲) before this complex with a much longer PR interval (0.36 sec) (↔). Immediately following this premature complex there is a narrow QRS complex tachycardia at a rate of 130 bpm. There are no P waves before these QRS complexes, but there are negative P waves after the QRS complexes with a stable RP interval (0.16 sec) (*). These QRS complexes, the negative P waves, and the RP interval are the same as what is seen in ECG 52A. This tachycardia is initiated by a premature atrial complex that has a PR interval that is much longer than that associated with the sinus complex. This is the usual way atrioventricular nodal reentrant tachycardia is initiated. Hence the mechanism for this tachycardia is an uncommon variant of a typical or common atrioventricular nodal reentrant tachycardia; this variant is known as slow-slow.

The anatomic basis for an atrioventricular nodal reentrant tachycardia is dual AV nodal pathways. One pathway conducts rapidly but recovers slowly (has a long refractory period). The second pathway conducts slowly, but recovers more quickly (has a short refractory period). These two pathways are proximally linked within the proximal junction and distally within the distal junction. During sinus rhythm the fast pathway predominates and conducts the impulse to the ventricles. If there is a premature atrial complex that reaches the AV node before the fast pathway has recovered, conduction is shifted to the slow pathway (which recovers more quickly); as a result of the slow conduction through this pathway, the PR interval is long. If the impulse via the slow pathway reaches the distal portion of the circuit at a time when the fast pathway has recovered, the impulse enters this pathway and is rapidly conducted retrogradely to the atrium, simultaneous with the antegrade conduction via the His-Purkinje system to the ventricles. If the impulse arrives at the proximal portion of the circuit when the slow pathway has recovered, the impulse will again be conducted antegradely via this pathway to the ventricle. If this process continues, an atrioventricular nodal reentrant tachycardia is established. As there is simultaneous activation of the atria (retrogradely) and ventricles (antegradely) the P wave occurs during the QRS complex, resulting in a no RP tachycardia. This is known as slow-fast. However, a variant of this typical AVNRT occurs when the fast pathway conducts relatively slowly, as a result of age-related changes in the pathway or the effect of a drug. In this situation, since the fast retrogradely conducting pathway is relatively slow in conduction, a short-PR tachycardia occurs, as is the case with this patient.

continues

Podrid's Real-World ECGs



ECG 52C Analysis: Sinus tachycardia

ECG 52C is from the same patient as ECG 52A and 52B. There is a regular rhythm at a rate of 110 bpm. The QRS complex duration, morphology, and axis are the same as in ECGs 52A and 52B. The QT/QTc intervals are also the same. There are P waves (+) before each QRS complex with a stable PR interval (0.18 sec). The P waves are

positive in leads I, II, aVF, and V4–V6. Hence this is a sinus tachycardia. Importantly, the QRS complexes and ST segments in this ECG can be compared to those in ECGs 52A and 52B, again confirming that the negative waveforms after the QRS complexes in these leads, which are not present in this ECG, are indeed negative P waves. ■

Notes

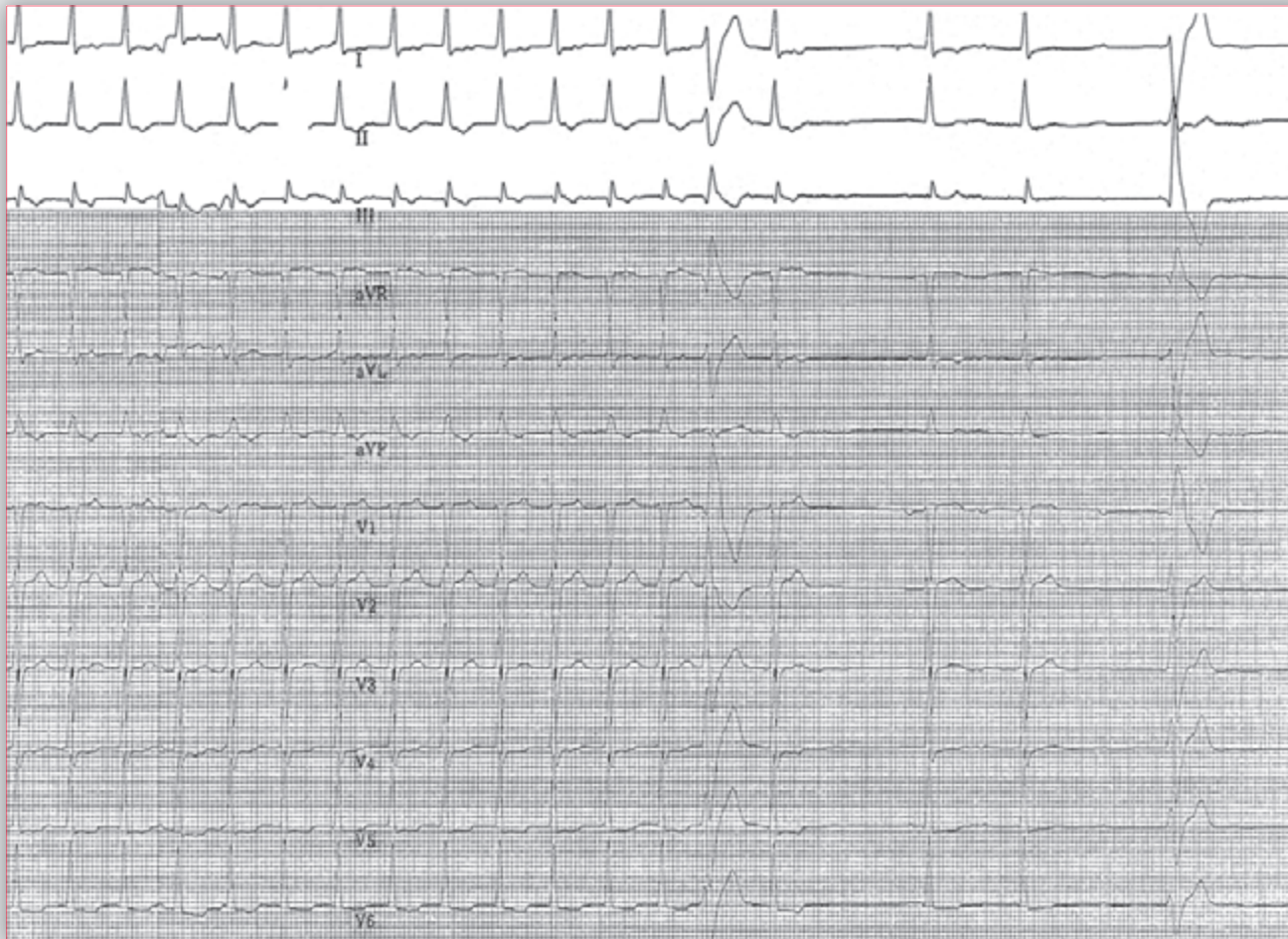
Core Case 53

You admit a 56-year-old patient to the medical floor after a prolonged stay in the emergency department, during which time he was treated for a narrow complex tachycardia. Your colleague discusses the case with the emergency department physician while you go

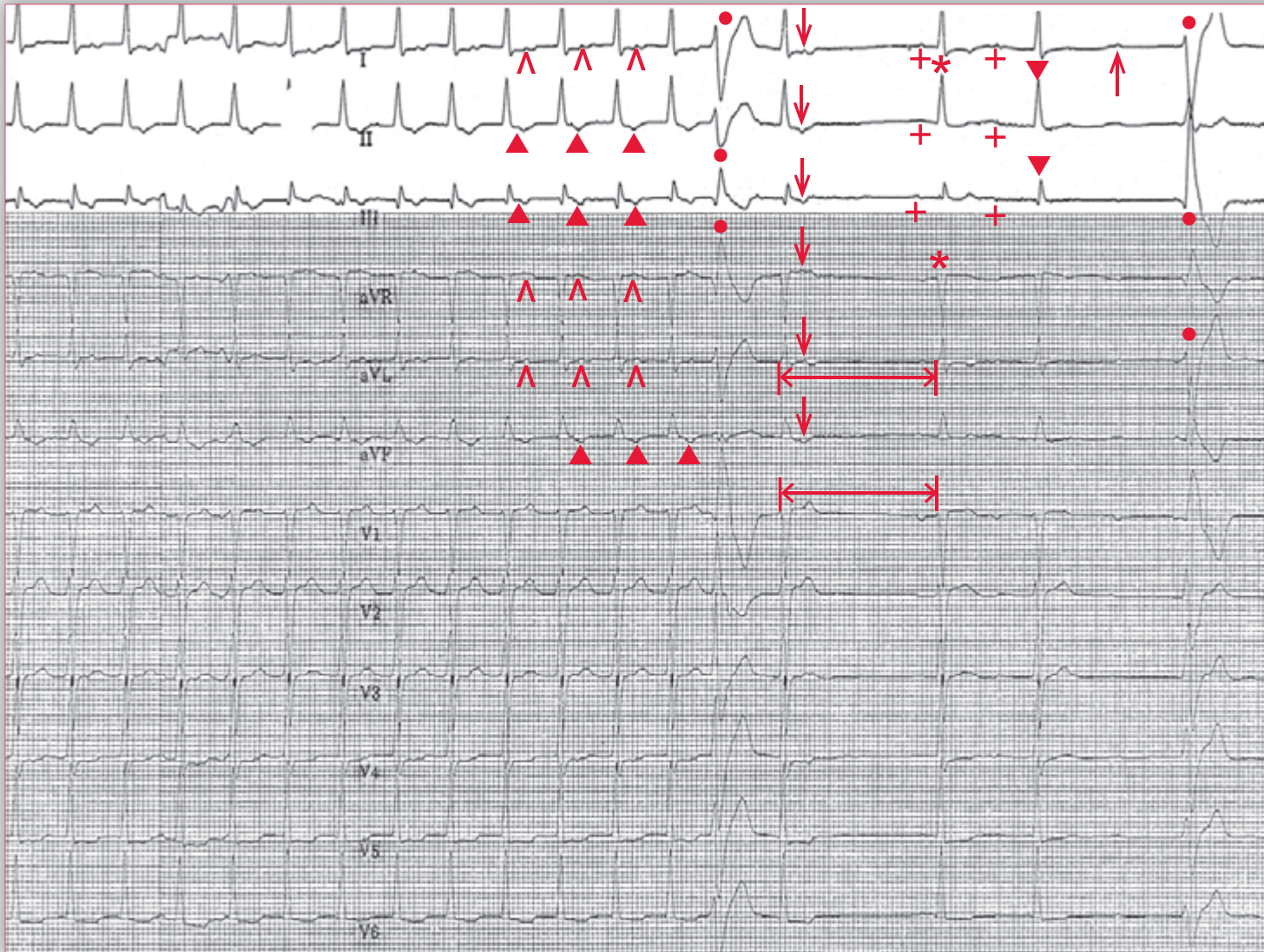
through the medical record. When you enter the patient's room, he is complaining of recurrent palpitations. An ECG is obtained, and it again shows a narrow complex tachycardia. You decide to repeat the therapy given in the emergency department.

What does his ECG show?

What treatment could account for the phenomenon observed on the ECG?



Podrid's Real-World ECGs



ECG 53 Analysis:
 Narrow complex
 supraventricular
 tachycardia (short-RP
 tachycardia:
 atrioventricular nodal
 reentrant tachycardia
 vs. atrioventricular
 reentrant tachycardia),
 acute termination to
 sinus bradycardia with
 Mobitz type I second-
 degree AV block
 (Wenckebach), and a
 ventricular escape beat

ECG 53 initially shows a regular rhythm at a rate of 140 bpm. The QRS complexes have a normal duration (0.08 sec) and morphology. The axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (300/460 msec).

There are no P waves seen before any of the QRS complexes. However, there are notches within the ST segments seen in leads I, aVR, and aVL (\wedge), which are suggestive of superimposed P waves. In addition, there are negative waveforms after the QRS complex in leads II, III, and aVF (\blacktriangle). This is, therefore, a short-RP (long PR) tachycardia. The RP interval is 0.20 second and the PR interval is 0.26 second. Etiologies for a short-RP tachycardia include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, an ectopic junctional tachycardia, an atrioventricular reentrant tachycardia (AVRT), or a typical or common atrioventricular nodal reentrant tachycardia (AVNRT) of an unusual variant (*ie*, slow-slow). The P waves are negative in leads II, III, and aVF, and hence this is not a sinus tachycardia. In addition, there is no evidence for a second atrial waveform, making atrial flutter unlikely. Etiologies include an atrial tachycardia or an arrhythmia involving the AV node.

After the fifteenth QRS complex, there is a pause (\leftrightarrow). Following the pause, the rate is slowed and the QRS complexes have the same duration, morphology, and axis as present before the pause. However, there are now P waves seen before the QRS complexes ($+$). The P waves are positive in leads I, II, aVF, and V4–V6. Hence these are sinus complexes. The first QRS complex after the pause (complex 16) ($*$) has a PR interval of 0.20 sec, while the next QRS complex (complex 17) (\blacktriangledown) has a PR interval of 0.32 second. There is a nonconducted P wave after the

seventeenth QRS complex (\uparrow). This is a second-degree heart block with a pattern of Mobitz type I second-degree AV block or Wenckebach. When the QRS complex morphology during the tachycardia is compared to the sinus QRS complexes after the tachycardia terminates, it can be seen that the notches and negative Q waves within the ST segment are not seen with the sinus QRS complex. Hence these are indeed negative P waves that follow the QRS complex.

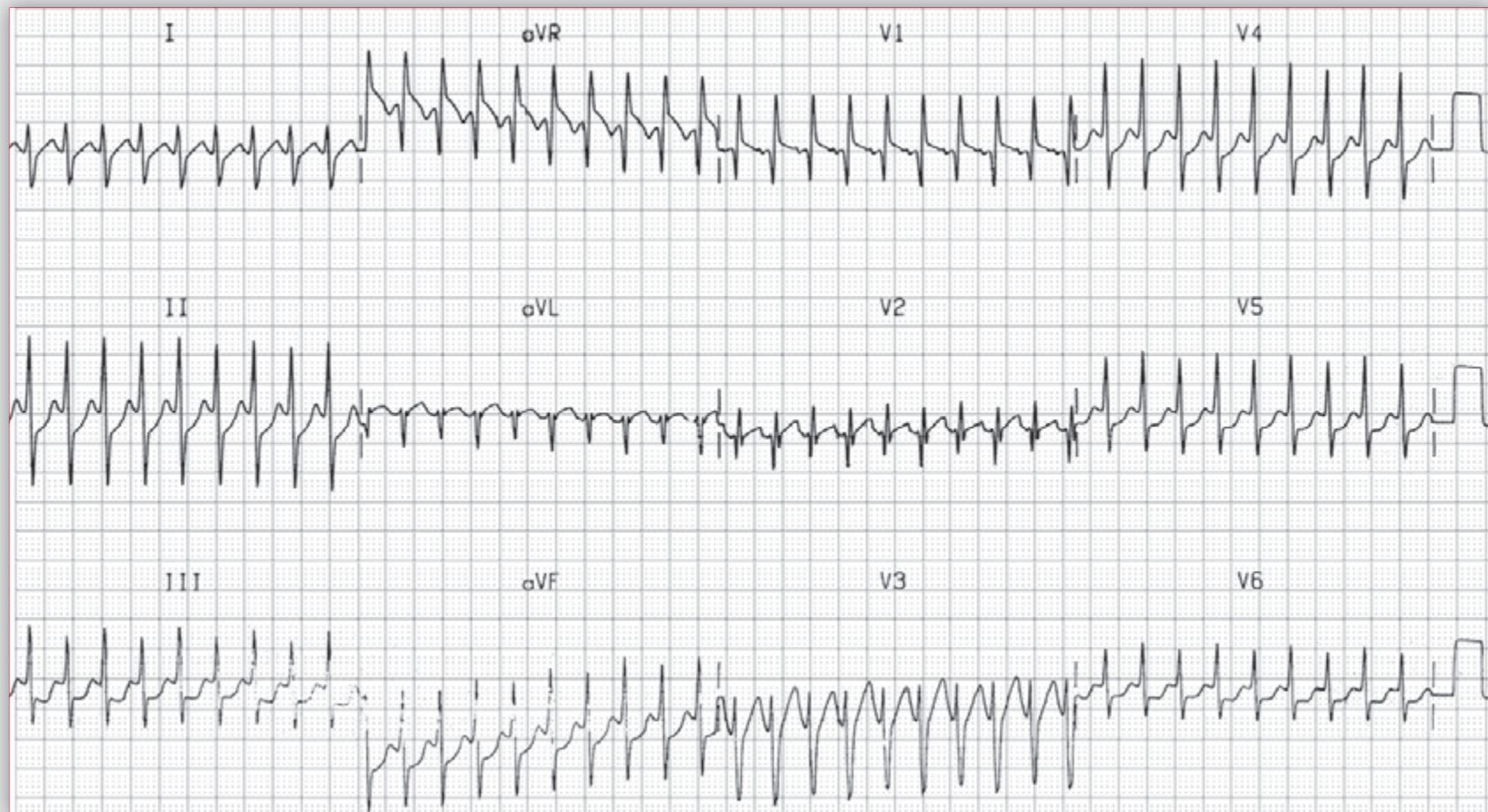
It can be noted that the last QRS complex of the tachycardia has a notching of the ST segment in leads I, aVR, and aVL and the negative P wave in leads II, III, and aVF (\downarrow). Therefore, the tachycardia terminates with a nonconducted P wave. Arrhythmias originating in the AV node or junction terminate with a nonconducted P wave. A retrograde P wave is still generated before the junctional mechanism terminates abruptly. Atrial arrhythmias terminate with the absence of a P wave as the atrial focus abruptly stops, ending the arrhythmia. This arrhythmia is either an ectopic junctional tachycardia, AVNRT, or AVRT. The arrhythmia terminated as a result of adenosine, making either an AVNRT or AVRT the likely mechanism. As there is no evidence for preexcitation on the ECG, an AVRT in this case would be the result of a concealed bypass tract. The acute termination of the tachycardia shortly and the development of transient Wenckebach after the termination of the arrhythmia is often seen with the use of adenosine.

Additionally, there are two wide QRS complexes seen (\bullet). The first one (complex 14) is premature, *ie*, a premature ventricular complex. Note that it does not terminate the tachycardia. The last QRS complex has the same morphology. However, it occurs after a pause. This is an escape ventricular complex. ■

Core Case 54

A 26-year-old woman has a history of intermittent palpitations. She presents to an emergency department shortly after the occurrence of an episode of palpitations. An ECG is obtained (ECG 58A). What are the potential etiologies for the arrhythmia? Following therapy, the tachycardia abruptly terminates and an ECG is recorded (ECG 58B).

ECG 54A

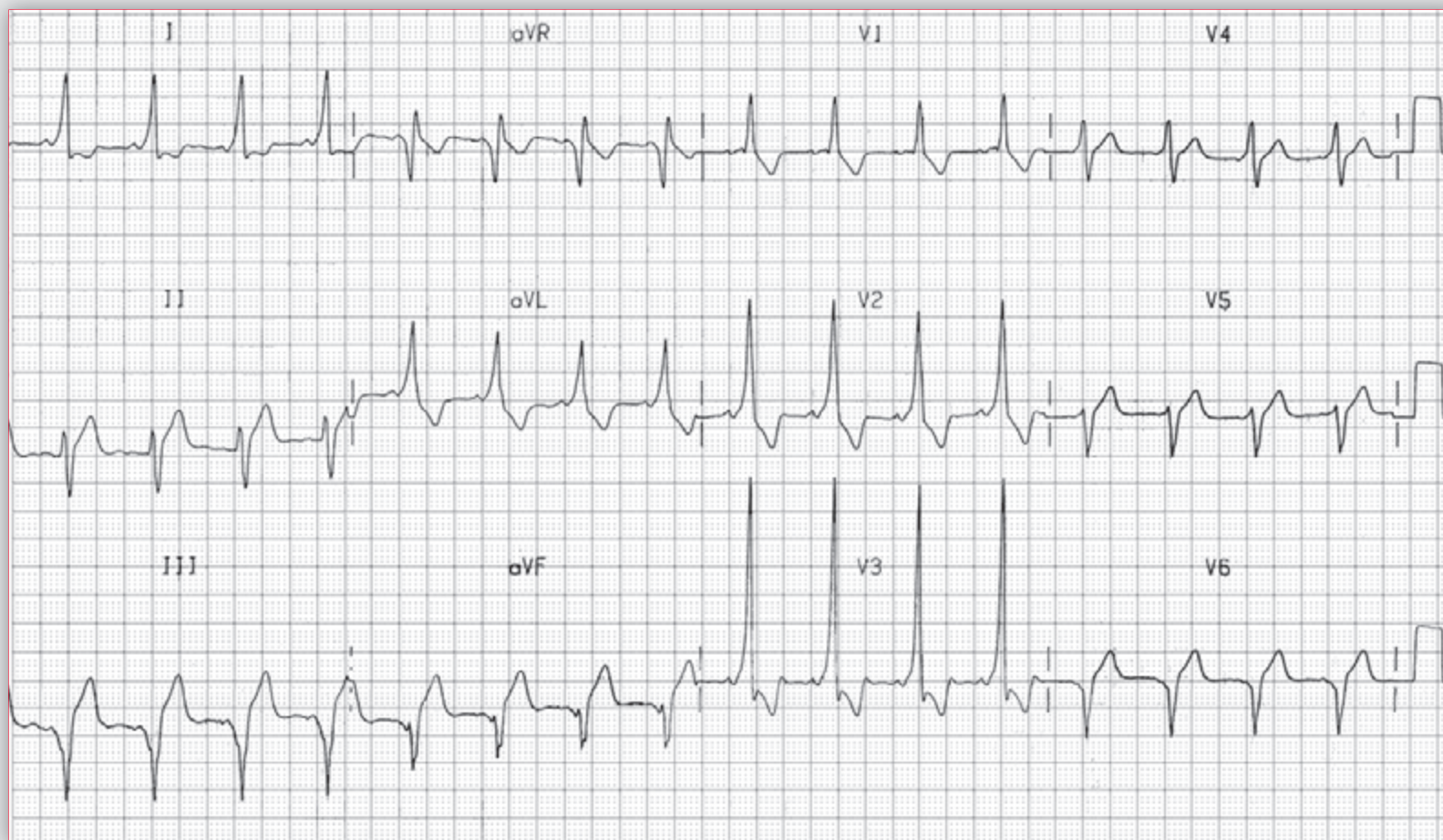


What abnormality is seen?

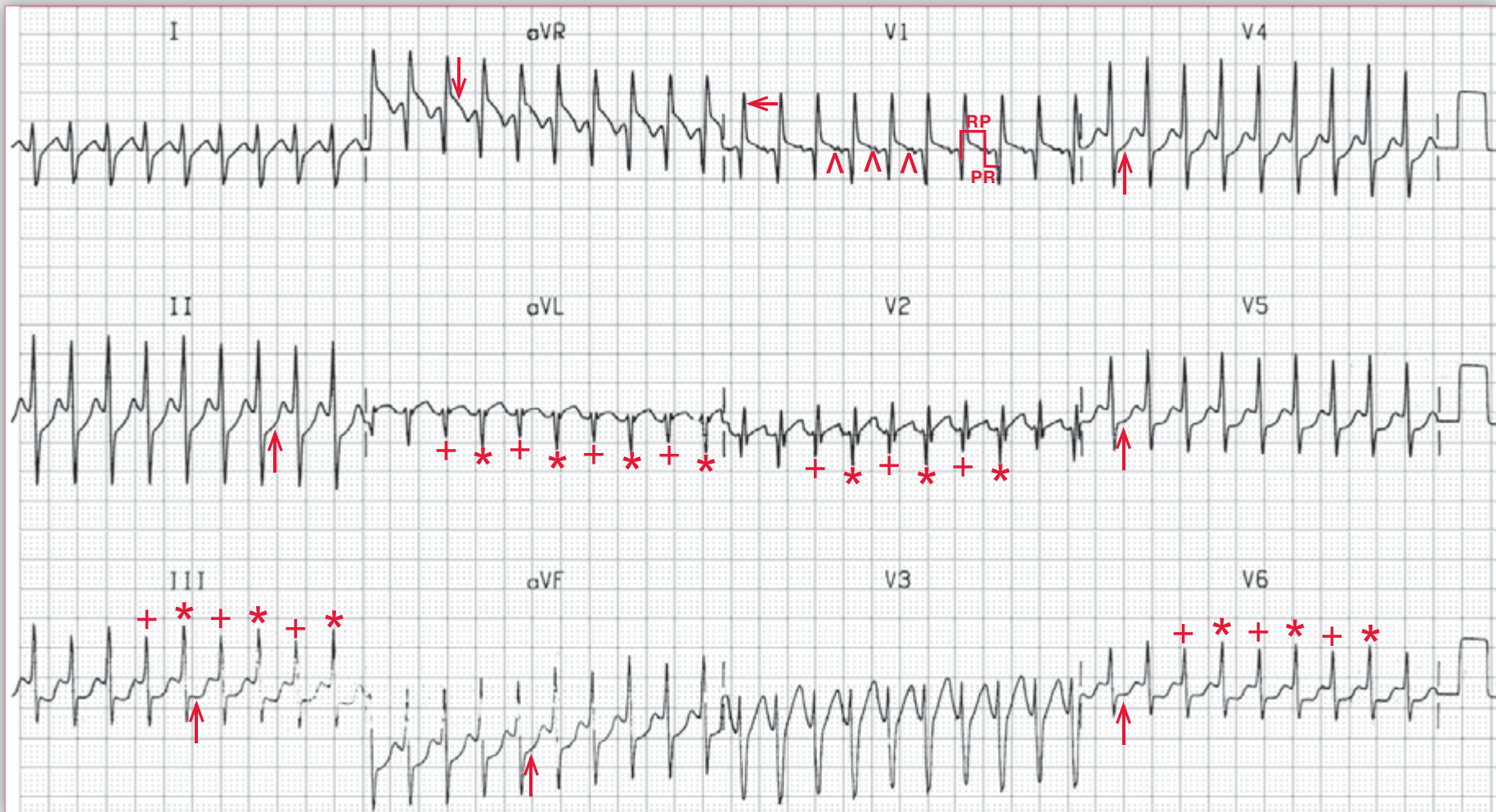
What is the likely etiology of the arrhythmia?

What therapy is effective?

ECG 54B



Podrid's Real-World ECGs



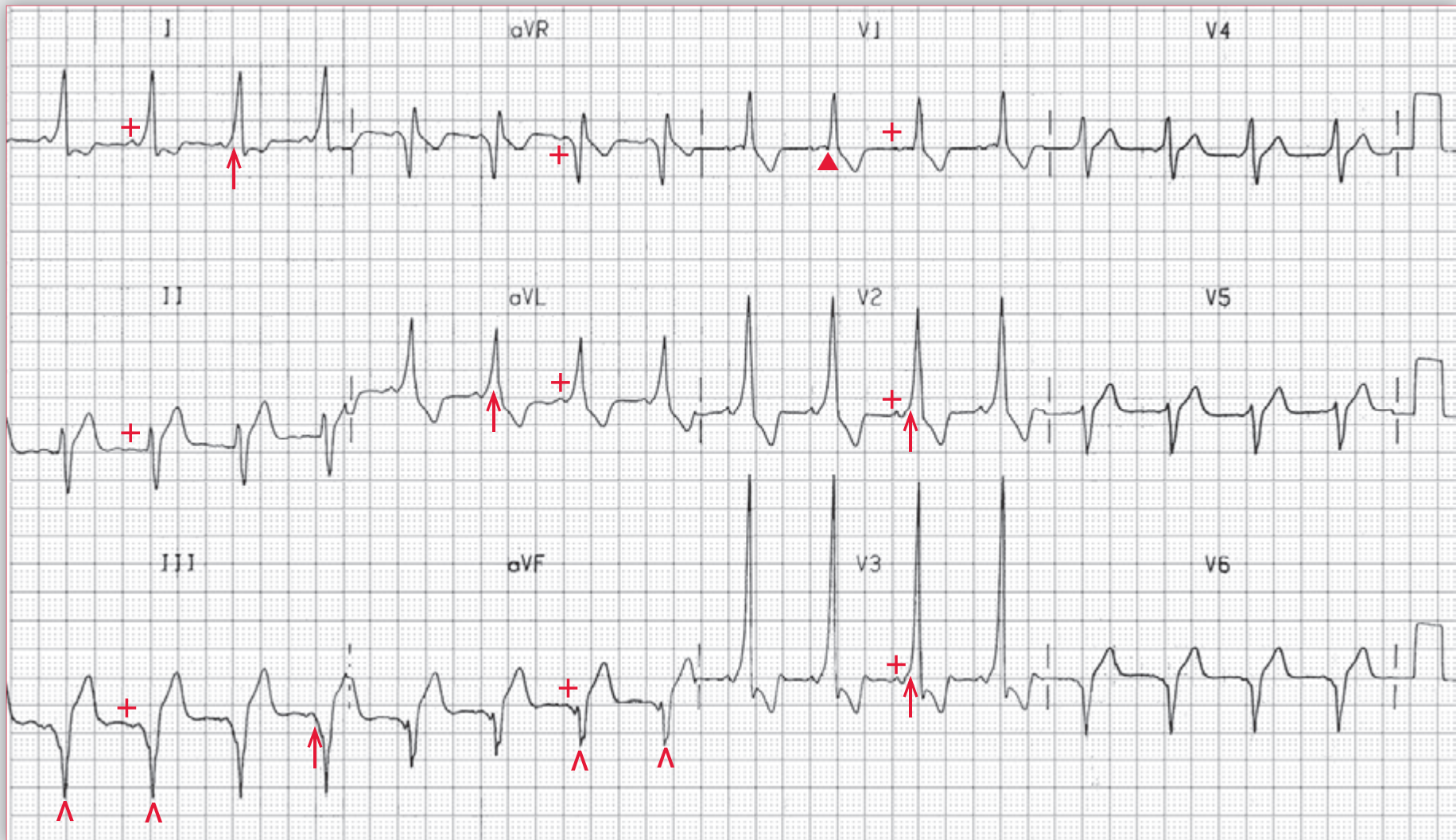
ECG 54A Analysis: Narrow complex supraventricular tachycardia (long-RP tachycardia), orthodromic atrioventricular reentrant tachycardia, right ventricular conduction delay, upsloping ST-segment depression electrical (QRS) alternans

ECG 54A shows a regular rhythm at a rate of 240 bpm. The QRS complex duration is normal (0.08 sec). The axis is rightward between $+90^\circ$ and $+180^\circ$ (QRS complex negative in lead I and positive in lead aVF). There is an R' in lead V1 (\leftarrow), consistent with a right ventricular conduction delay (also termed a crista pattern). The QT/QTc intervals are normal (220/440 msec). There are upsloping ST-segment depression in leads II, III, aVF, and V4–V6 (\uparrow). There is ST segment elevation (\downarrow) in lead aVR, which is actually ST segment depression. Although there are no obvious P waves seen before or after any QRS complex, there is notching seen before the QRS complex in lead V1 (\wedge). These are likely P waves and hence this is a long-RP tachycardia (RP interval = 0.20 sec

and PR interval = 0.12 sec). The etiologies include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, an uncommon or atypical atrioventricular nodal reentrant tachycardia or an atrioventricular reentrant tachycardia. The ECG does not provide any additional features that can establish the etiology. Also noted is QRS or electrical alternans, *ie*, beat-to-beat changes in QRS amplitude best seen in leads III, aVL, V2, and V6 (\pm ,*). Electrical or QRS alternans can be seen with any rapid supraventricular tachycardia and is the result of beat-to-beat changes in calcium fluxes into the myocardium.

continues

Podrid's Real-World ECGs



ECG 54B Analysis: Normal sinus rhythm, Wolff-Parkinson-White pattern, pseudo inferior wall myocardial infarction

ECG 54B shows a regular rhythm at a rate of change to 98 bpm. There is a P wave (+) before each QRS complex with a short but constant PR interval (0.10 sec). The P wave is positive in leads I, II, aVF and V4–V6; hence this is a normal sinus rhythm. The QRS duration is prolonged (0.16 sec) and there is a prominent slurring of the upstroke of the QRS complex (↑), known as a delta wave. These features are characteristic of a Wolff-Parkinson-White pattern. In this condition, there is an accessory pathway or bypass tract (bundle of Kent) that serves as a second AV connection, along with the normal AV node–His-Purkinje system. The atrial impulse is conducted via both pathways, but since the accessory pathway bypasses the normal AV node, which is the part of the electrical system that has the slowest rate of conduction, the PR interval is short. There is direct ventricular myocardial activation initiated via the accessory pathway, bypassing the normal His-Purkinje system. Since impulse conduction via the myocardium is slower than via the His-Purkinje system, the onset of the QRS complex is early (preexcited) and slow, accounting for the slurred upstroke or the delta wave. There is a Q wave in leads III and aVF (Λ), which represents a pseudo-infarction pattern of the inferior wall that is associated with a

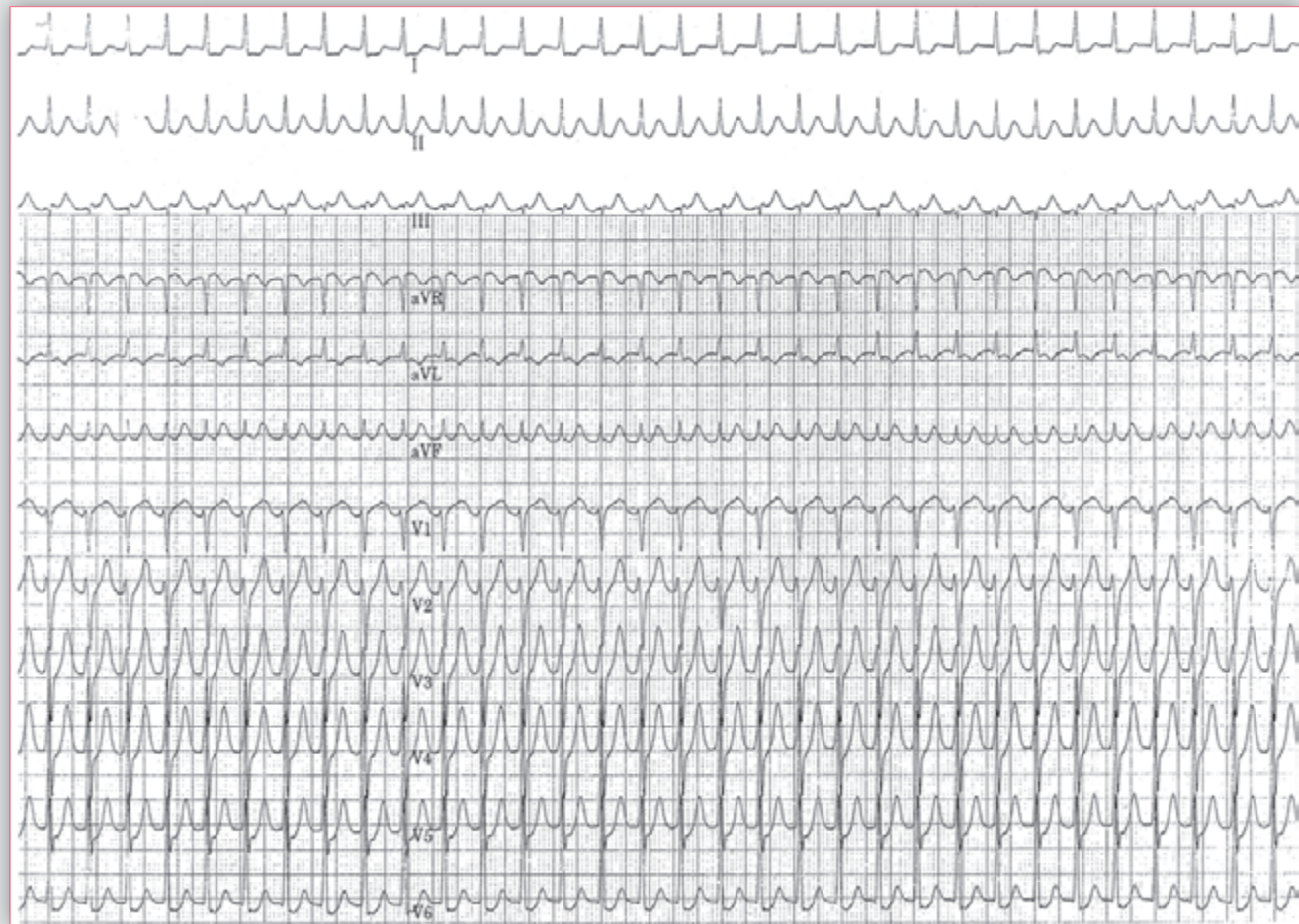
posteroseptal bypass tract. The delta wave is positive in lead V1 (▲), termed type A, consistent with a left-sided bypass tract. Hence the arrhythmia in ECG 54A is an atrioventricular reentrant tachycardia (AVRT). The presence of narrow QRS complexes indicates that this is an orthodromic AVRT, *ie*, the antegrade activation of the ventricles is via the normal AV node–His-Purkinje system and the retrograde activation of the atria is via the accessory pathway. Orthodromic AVRT as the etiology can only be established after the arrhythmia is terminated, demonstrating sinus complexes that are preexcited.

Termination of an AVRT usually involves altering conduction through the AV node, which is a part of the circuit and is the structure with slowest conduction through it. However, interruption of any part of the reentrant circuit will result in arrhythmia termination. When the AVRT is orthodromic, with a narrow QRS complex, there is no issue with the use of an AV nodal blocking agent for arrhythmia termination, as ventricular activation occurs via the normal AV node–His-Purkinje system and is clearly supraventricular. ■

Core Case 55

An 18-year-old man is seen three times in the emergency department because of palpitations. On his first two visits, ECG 55A and ECG 55B are recorded, but a diagnosis cannot be definitively made based on ECG alone.

ECG 55A



On the third visit, the recorded ECG shows a different arrhythmia (ECG 55C), but a diagnosis is now established.

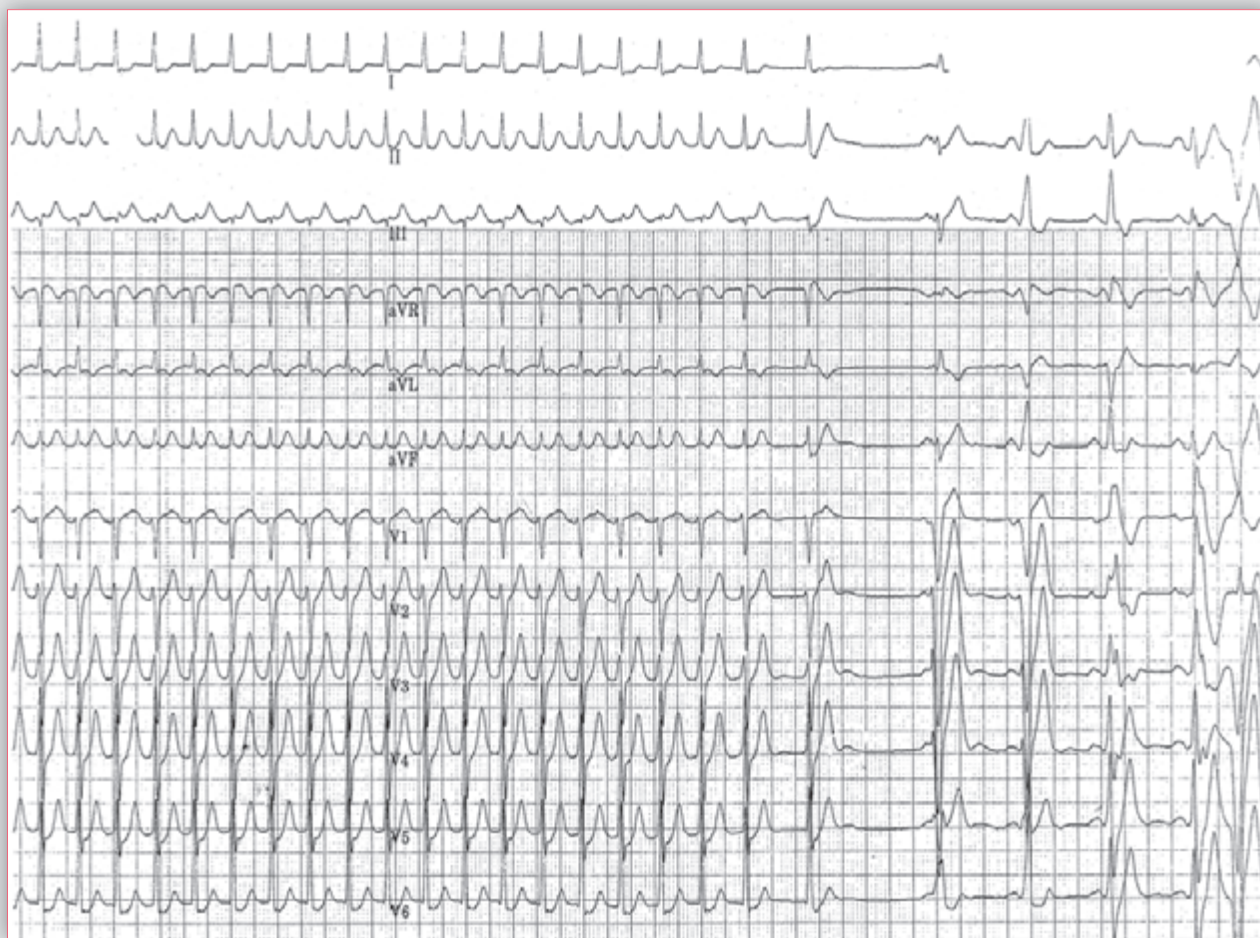
After appropriate therapy the arrhythmia is terminated and ECG 55D is recorded.

What arrhythmia is seen, and what is the underlying problem?

How should this arrhythmia be treated?

What abnormality is now definitively established?

ECG 55B

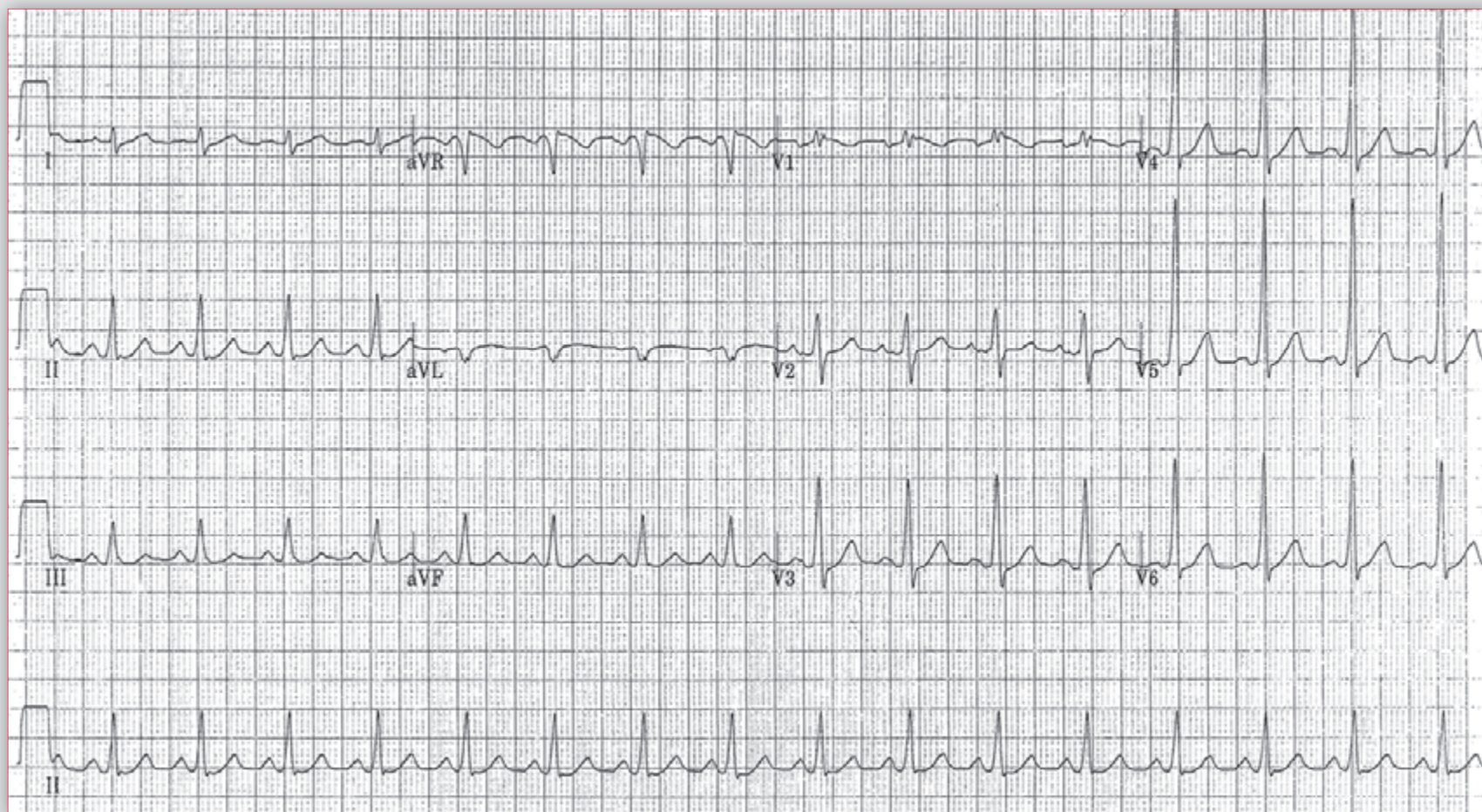


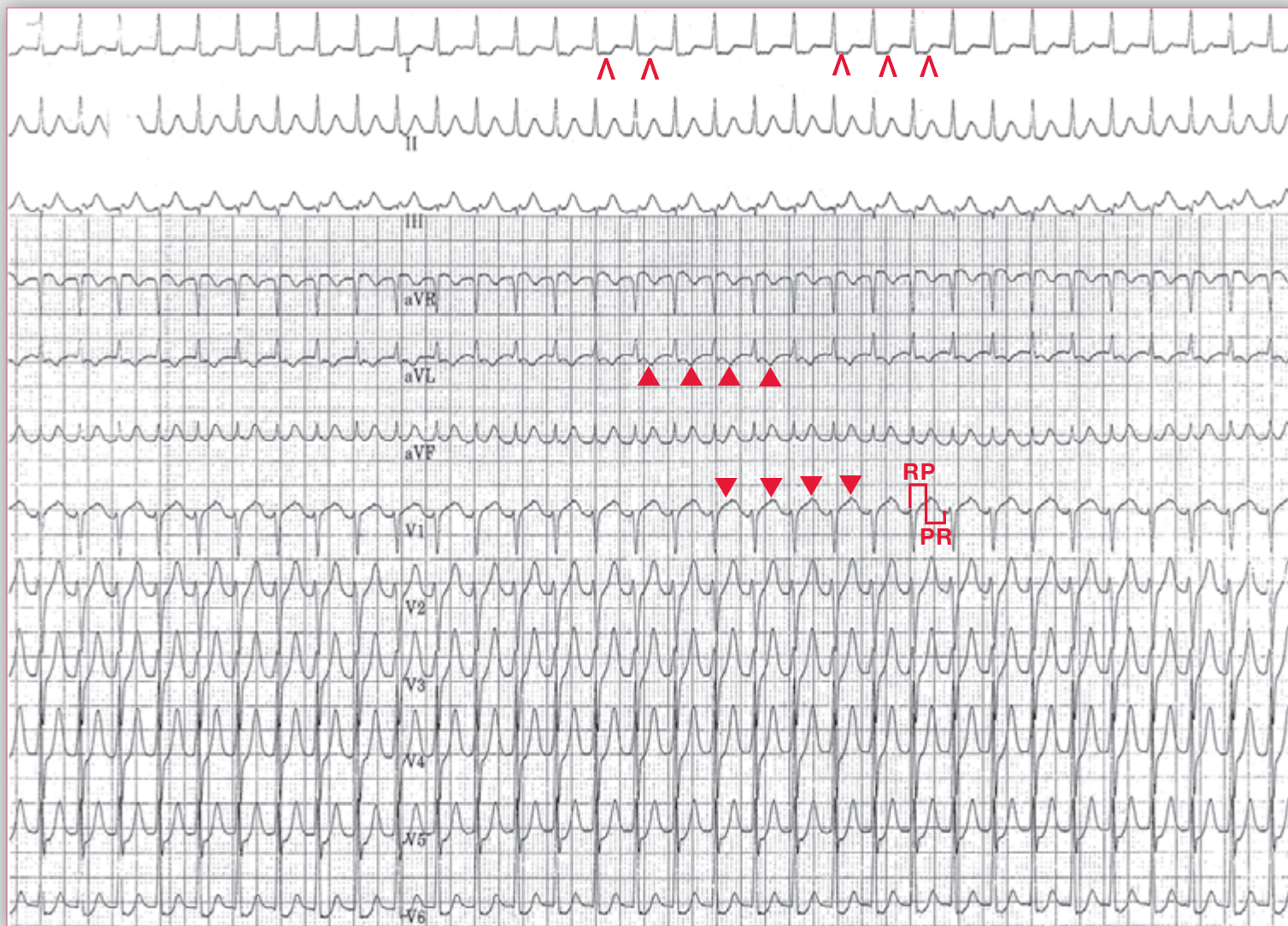
Core Case 55

ECG 55C



ECG 55D



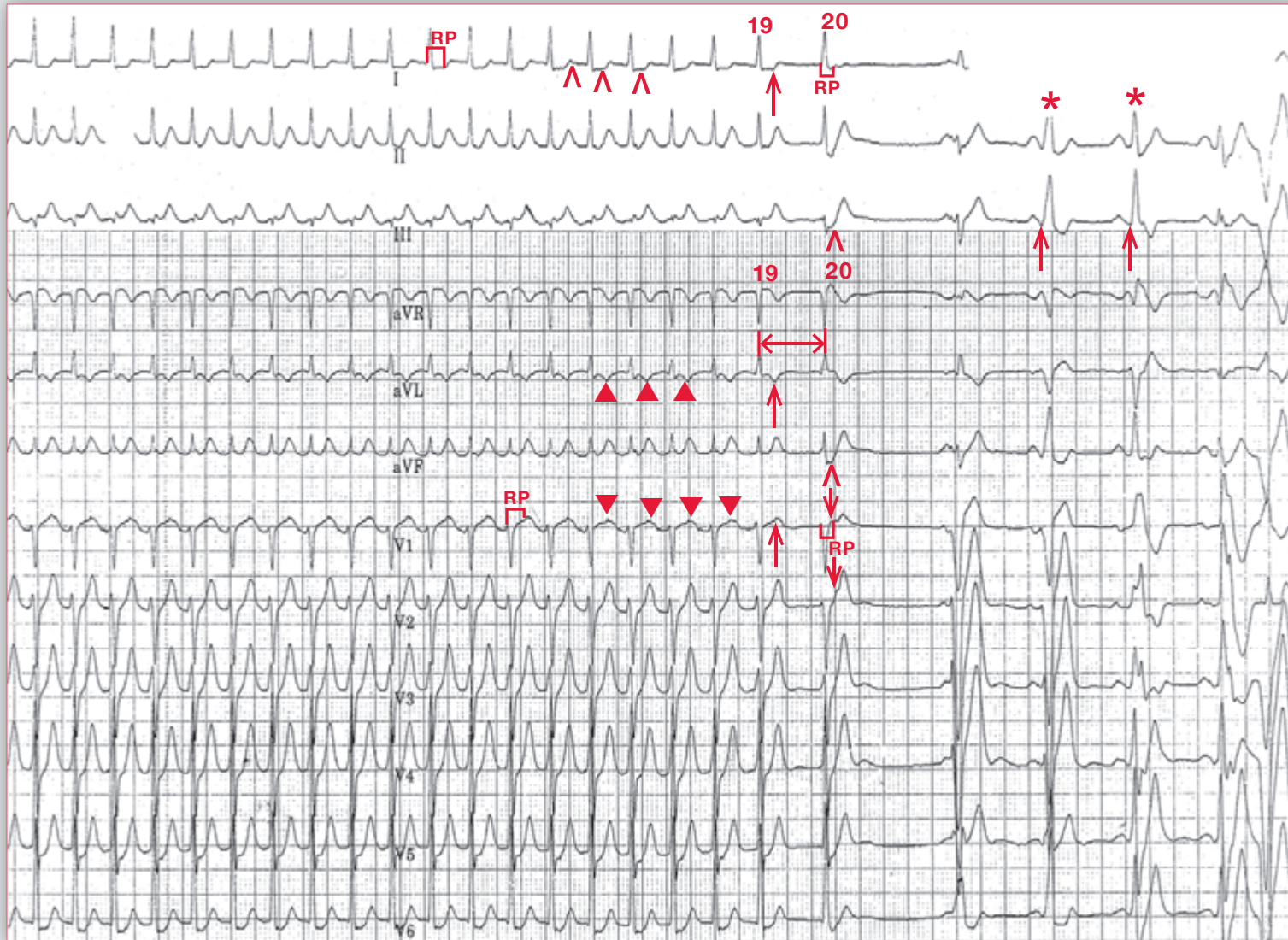


ECG 55A Analysis: Narrow complex supraventricular tachycardia, short PR tachycardia, orthodromic atrioventricular reentrant tachycardia

ECG 55A shows a regular rhythm at a rate of 220 bpm. The QRS complex duration is normal (0.08 sec), and there is a normal morphology and axis between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are slightly prolonged (240/460 msec). There are no obvious P waves seen before or after any of the QRS complexes. However, there are notches of the ST segment seen in lead I (^) and notches of the upstroke of the T wave (▼) noted in lead V1. There are also notches in lead aVL (▲), which have the same relationship to the QRS complex. They are regular and have a constant RP interval (0.18 sec) while the PR interval is 0.22 second. Hence this is a short-RP tachycardia. Etiologies for a short-RP tachycardia are

sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atrioventricular reentrant tachycardia (AVRT), and an unusual form of a typical atrioventricular nodal reentrant tachycardia (AVNRT), known as slow-slow. Typical AVNRT, which is due to dual AV nodal pathways, is termed slow-fast, *ie*, the slow pathway conducts antegrade to the ventricle while the fast pathway conducts retrograde to the atria. Hence there is no P wave seen as there is simultaneous atrial and ventricular activation. With slow-slow AVNRT the fast pathway conducts relatively slow, producing a short-RP tachycardia. The ECG and clinical history do not provide any additional clues in regard to the etiology of this tachycardia.

continues



ECG 55B Analysis: Narrow complex supraventricular tachycardia, short-PR tachycardia, acute termination to normal sinus rhythm, orthodromic atrioventricular reentrant tachycardia

ECG 55B is from the same patient as ECG 55A. There is a narrow complex tachycardia that is identical to that seen in ECG 55A. Hence it is a short-RP tachycardia. Notches in the ST segment are again seen in lead I (^), on the upstroke of the T wave in V1 (▼) and in aVL (▲). The RP interval (□) is the same as seen in ECG 55A. The arrhythmia terminates abruptly after the nineteenth QRS complex (↔). Noted is that after the nineteenth QRS complex, there the same abnormality in the ST segment and T waves as seen during the tachycardia (↑). Hence the tachycardia terminates with a retrograde P wave that is nonconducted. A supraventricular tachycardia that terminates with a nonconducted retrograde P wave originates within the AV node (*ie*, AVNRT or ectopic junctional tachycardia) or involves the AV node as an essential part of the circuit (*ie*, AVRT). In contrast, atrial arrhythmias terminate with the absence of atrial activity, *ie*, the atrial mechanism ceases and hence there is no P wave. Therefore, the etiology of this tachycardia involves the AV node.

The first QRS complex after the pause (complex 20) has the same duration and morphology as the QRS complexes during the tachycardia. There is no P wave before this QRS complex, but there is a notching of the ST segment (↓), best seen in leads I and V1–V2 as well as what looks like an S wave in leads II and aVF (^); this is also a retrograde P wave. However, the RP interval (⊐) is shorter (0.14 sec) than that seen during the tachycardia (□) (0.18 sec). This is an escape junctional complex with a retrograde P wave. Another pause follows this complex, and thereafter there are sinus P waves seen at a rate of 90 bpm. The QRS complexes have an increased duration with two different morphologies and various PR intervals. The etiologies of these QRS complexes is not clear. However, several of the complexes (*) have a short PR interval and a slurred upstroke (↑), suggesting a delta wave and a Wolff-Parkinson-White pattern.

continues



ECG 55C Analysis: Atrial fibrillation, Wolff-Parkinson-White pattern

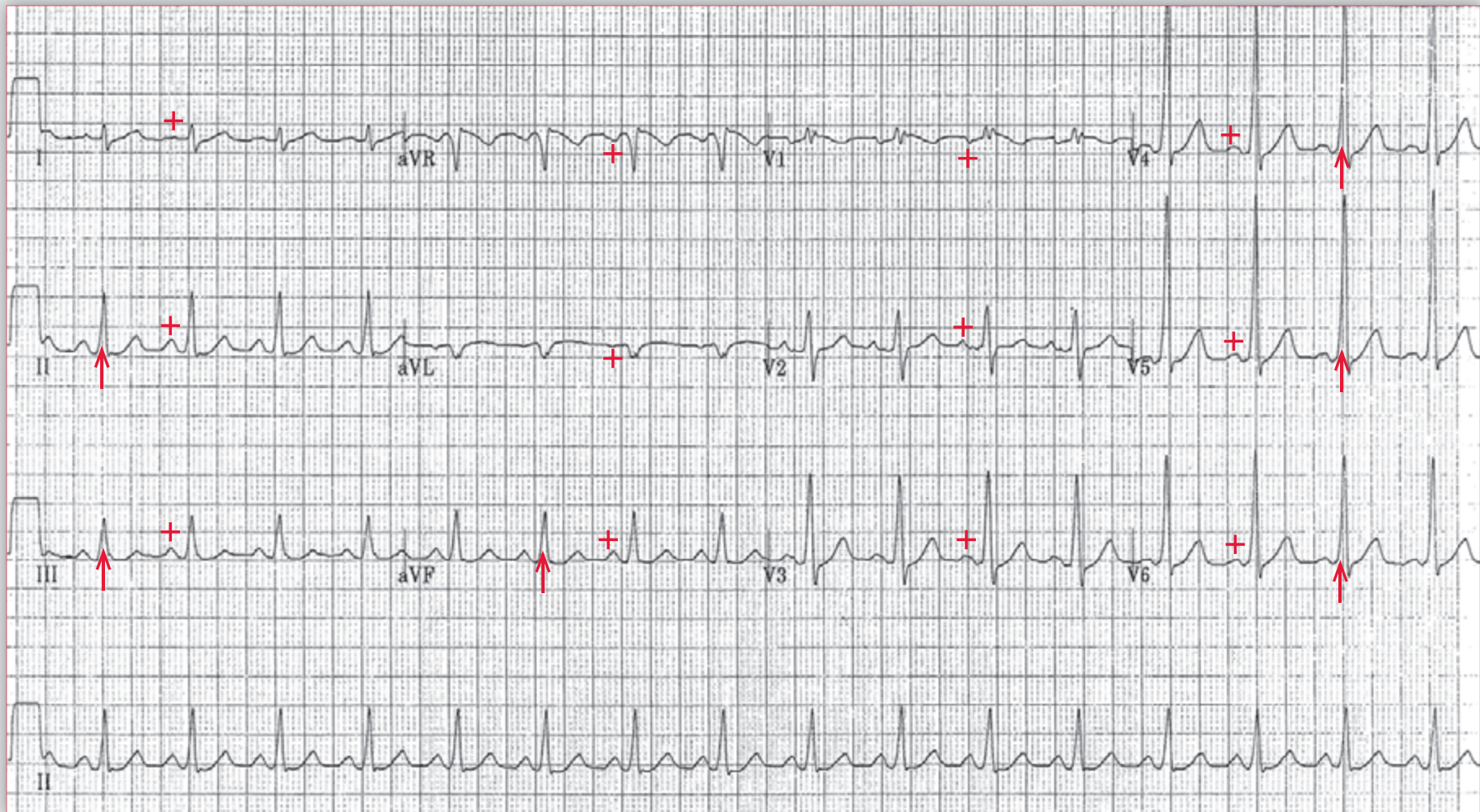
ECG 55C is from the same patient as ECG 55A and 55B. The rhythm is irregularly irregular, and there are no obvious P waves seen. There are only three rhythms that are irregularly irregular, and these include sinus arrhythmia in which there is only one P-wave morphology and a stable PR interval; multifocal atrial tachycardia (rate > 100 bpm) or multifocal atrial rhythm or wandering atrial pacemaker (rate < 100 bpm), in which there are > 3 different P-wave morphologies (without any dominant P-wave morphology) and PR intervals; or atrial fibrillation, in which there is no organized atrial activity. Hence the rhythm is atrial fibrillation. The QRS complexes have various durations and it can be seen that there is no relationship between QRS complex duration and rate. There are long RR intervals (⏏) that end with a wide QRS complex (*) and shorter RR intervals (⏏ associated with a narrow QRS complex (^). In addition there are differences in QRS complex width that are not associated with rate (↑) *ie*, wider complexes at slower rates and less wide at faster rates). These features are not seen with a rate-related aberration. With rate-related aberration the wide QRS complexes are seen with the faster rate, while the narrow QRS complexes are seen with the slower rate. The absence of any association between rate and QRS complex

width is typical of preexcitation, *ie*, Wolff-Parkinson-White (WPW) and not typical rate-related aberration. In addition, with a typical aberration (with a right or left bundle branch block), there is no change in QRS complex duration, as conduction through the His-Purkinje system remains constant regardless of the heart rate, *ie*, it is all or none. WPW is a fusion complex between conduction through the accessory pathway as well as through the normal AV node–His-Purkinje system. As a result of different degrees of fusion (which is related to the conduction properties of the AV node, which will determine how much ventricular activation is via the accessory pathway), the QRS complex duration can change unpredictably.

Importantly, the QRS complex morphology is similar to the QRS complex morphology of some of the QRS complexes in ECG 55B, which are preexcited. Since the patient has WPW, the etiology of the tachycardia in ECG 55A is most likely AVRT. As the QRS complex is narrow and normal in morphology, this is an orthodromic AVRT. Indeed, AVRT most often presents with a short-RP tachycardia, and one of the most common etiologies for a short-RP tachycardia is AVRT.

continues

Podrid's Real-World ECGs



ECG 55D Analysis: Normal sinus rhythm, Wolff-Parkinson-White pattern

ECG 55D is the baseline ECG for the patient with ECGs 55A, 55B, and 55C. There is a regular rhythm at a rate of 96 bpm. There is a P wave before each QRS complex (+) with a stable PR interval (0.14 sec). However, the P wave is broad, while the PR segment is very short. The QRS complex duration is increased (0.12 sec) and there is a slight slurring to the upstroke (†) seen in many of the leads; this is a delta wave. The QT/QTc intervals are slightly prolonged 360/455 msec but are normal when the increased QRS complex duration is considered (320/405 msec). The short PR segment, the widened QRS complex duration and the delta wave are characteristic of a WPW pattern. The QRS complex has a similar morphology to what was seen in ECG 55C, although not as wide. Hence some of the QRS complexes in ECG 55C are maximally preexcited (*ie*, all of ventricular activation is via the accessory pathway), while those in this ECG show more fusion between conduction via the normal AV node–His–Purkinje system and the accessory pathway.

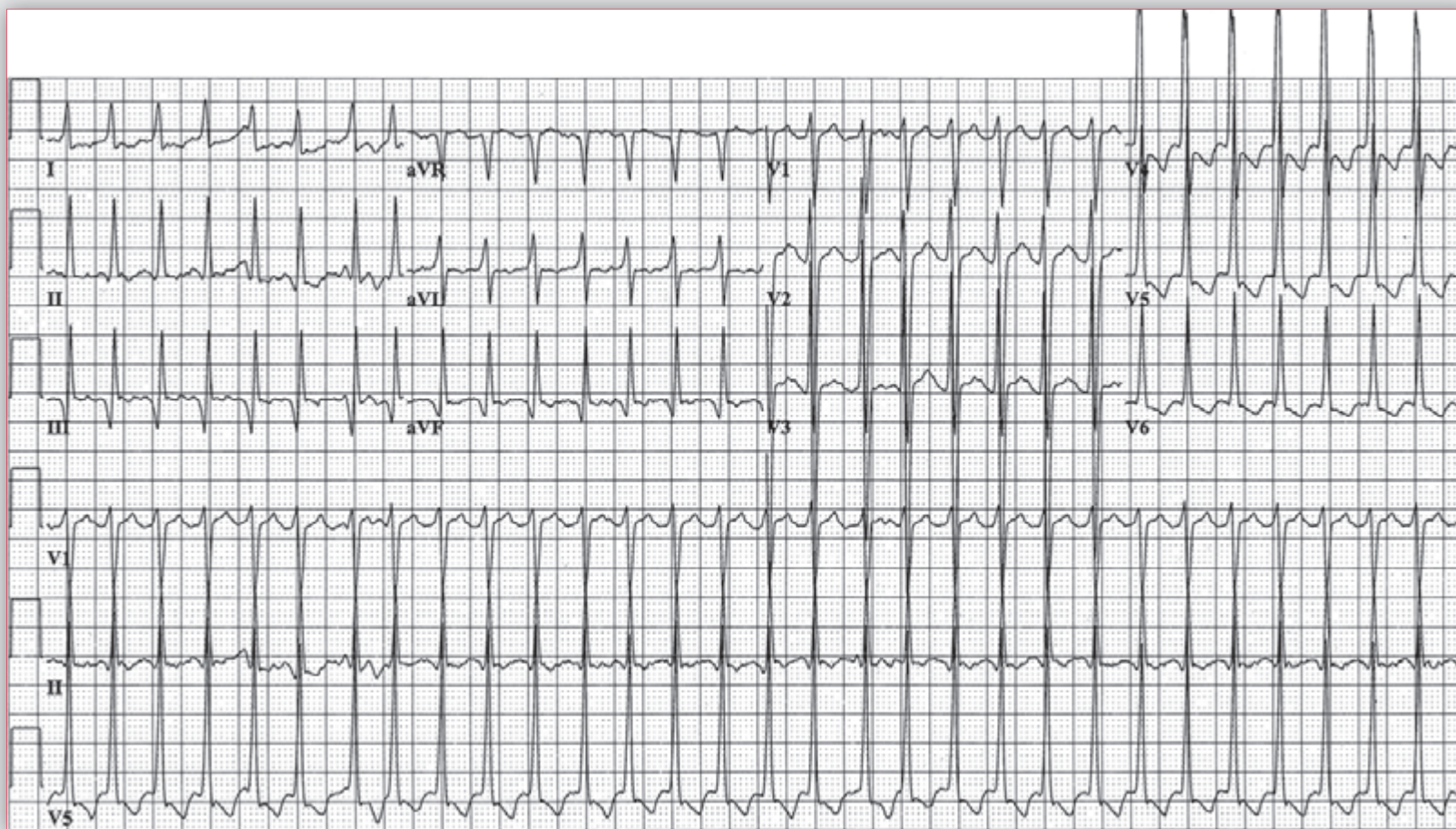
Termination of an AVRT involves altering conduction through the AV node that is a part of the circuit. Interruption of any part of the reentrant circuit will result in arrhythmia termination. When the AVRT is orthodromic, with a narrow QRS complex, there is no issue with the use of an AV nodal blocking agent for arrhythmia termination as ventricular activation occurs via the normal AV node–His–Purkinje system and is clearly supraventricular. It is likely that the patient received adenosine, which accounts for the variability in the PR intervals and QRS complexes after the arrhythmia terminated (ECG 55B).

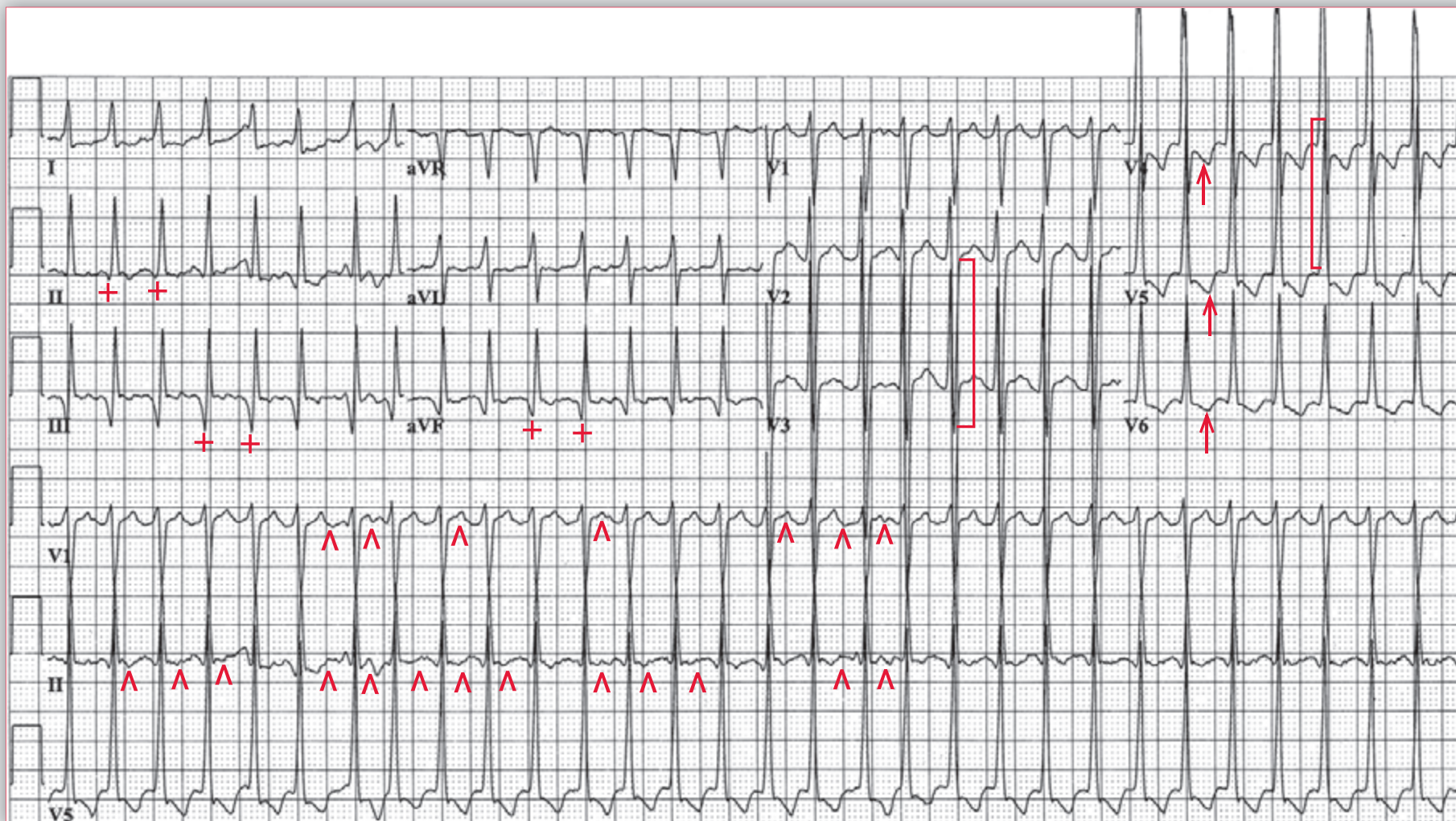
However, patients with WPW who have atrial fibrillation should not receive an AV nodal blocking agent, which is often given for rate control. In this situation, blocking the AV node will result in all of atrial activation via the accessory pathway, which is capable of rapid conduction as it generally has a short refractory period. Since the atrial rate in atrial fibrillation is in excess of 350 to 450 bpm, the short refractory period of the accessory pathway will allow for rapid impulse conduction and hence the ventricular rate may be very rapid in response to the rapid atrial rate. Ventricular rates of > 350 bpm may be seen, and at these rate ventricular fibrillation may be provoked, even in a structurally normal heart. It is the occurrence of atrial fibrillation and the precipitation of ventricular fibrillation that accounts for the sudden cardiac arrest seen in these patients. The drugs of choice for atrial fibrillation in WPW include IV procainamide and IV ibutilide. These drugs not only slow conduction through the accessory pathway (by slowing the rate of impulse conduction or prolonging refractoriness) but they also have the potential to revert AF to NSR. Clinically, lidocaine may be useful as it will also slow and possibly block conduction through the accessory pathway, resulting in a shift of impulse conduction only through the AV node–His–Purkinje system. In this situation, the QRS complex will narrow and become normal (supraventricular in morphology), although lidocaine will not terminate the AF. As conduction is no longer through the accessory pathway, this risk of very rapid ventricular rates and ventricular fibrillation is eliminated. For patients who have hemodynamic impairment, electrocardioversion is indicated. ■

Notes

A 58-year-old woman presents to her primary care physician with complaints of her heart racing. She has never experienced this sensation before and indeed has no known heart disease. She does have a history of hypertension, although it is not certain if her blood pressure has been controlled.

What new diagnosis can be made on the ECG?





ECG 56 Analysis: Atrial fibrillation, old inferior wall myocardial infarction, left ventricular hypertrophy with associated ST-T wave changes

ECG 56 shows an irregularly irregular rhythm (no pattern to the RR intervals) at an average rate of 184 bpm. When the rate is this rapid the RR intervals may appear to be regular as subtle differences in the RR intervals may not be apparent. Subtle differences may become more obvious if 2–3 intervals are measured at the same time. The QRS complex duration is normal (0.08 sec) and the axis is normal between 0° and $+90^\circ$ (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (240/420 msec). There are significant Q waves (+) in leads II, III, and aVF, diagnostic of an old inferior wall myocardial infarction. The amplitude of the QRS complex is increased in lead V2 (S wave = 29 mm) (]) and in lead V5 (R wave = 29 mm) ([) (S+R = 58 mm), meeting criterion for left ventricular hypertrophy (S+R \geq 35 mm). There are ST-T wave changes in V4–V6 (\uparrow), likely secondary to left ventricular hypertrophy.

There are no obvious P waves seen. There are irregular undulations of the baseline in between each RR interval (\wedge). These are fibrillatory waves and hence the rhythm is atrial fibrillation with a rapid ventricular response.

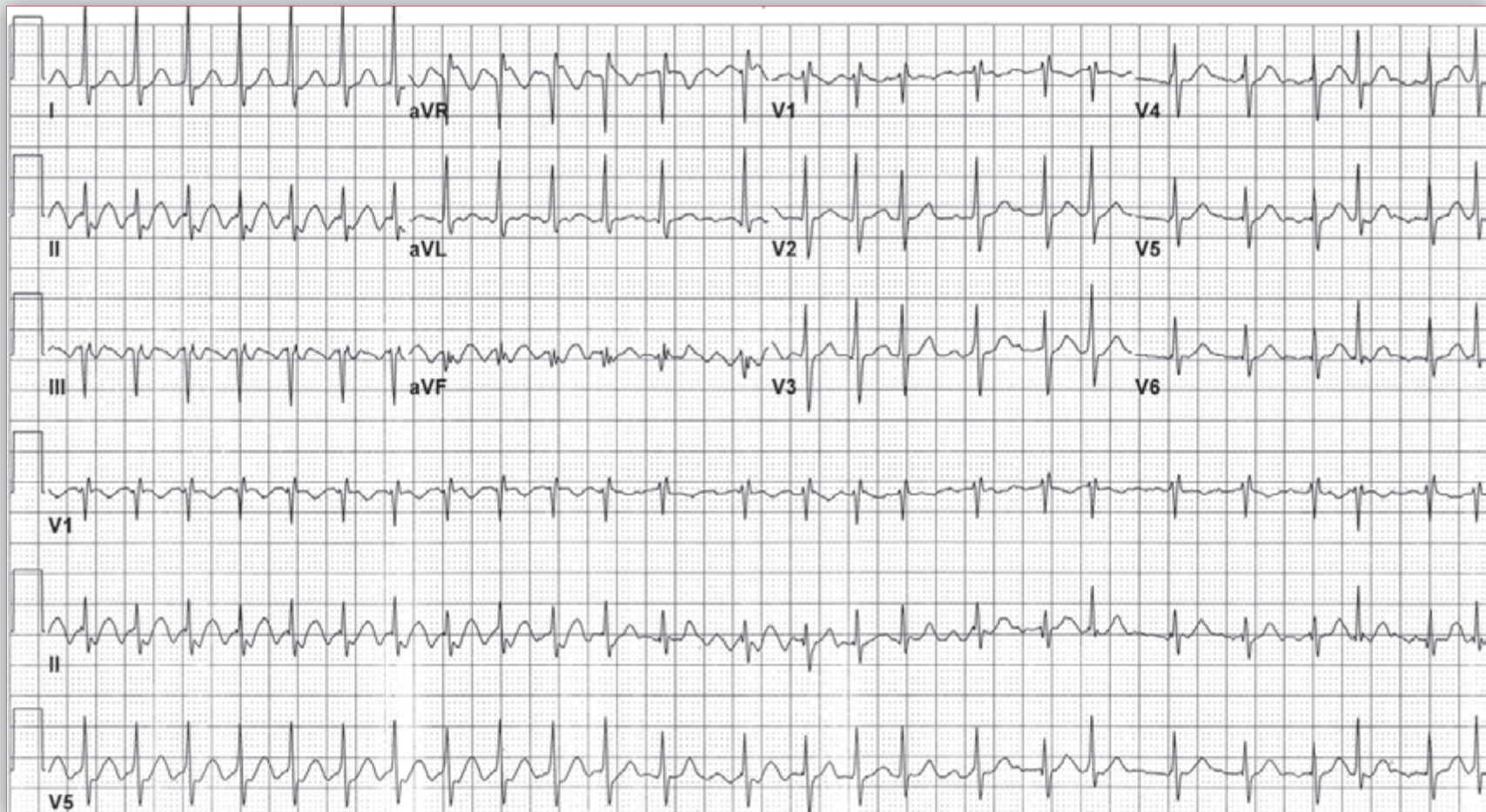
There are only three supraventricular rhythms that have RR intervals that are irregularly irregular. These include sinus arrhythmia (in which there is one P-wave morphology and a stable PR interval), multifocal atrial rhythm (rate < 100 bpm) or multifocal atrial tachycardia (rate > 100 bpm) (in which there are \geq 3 P-wave morphologies with no one P-wave morphology being dominant and variable PR intervals), and atrial fibrillation (in which there is no organized atrial activity).

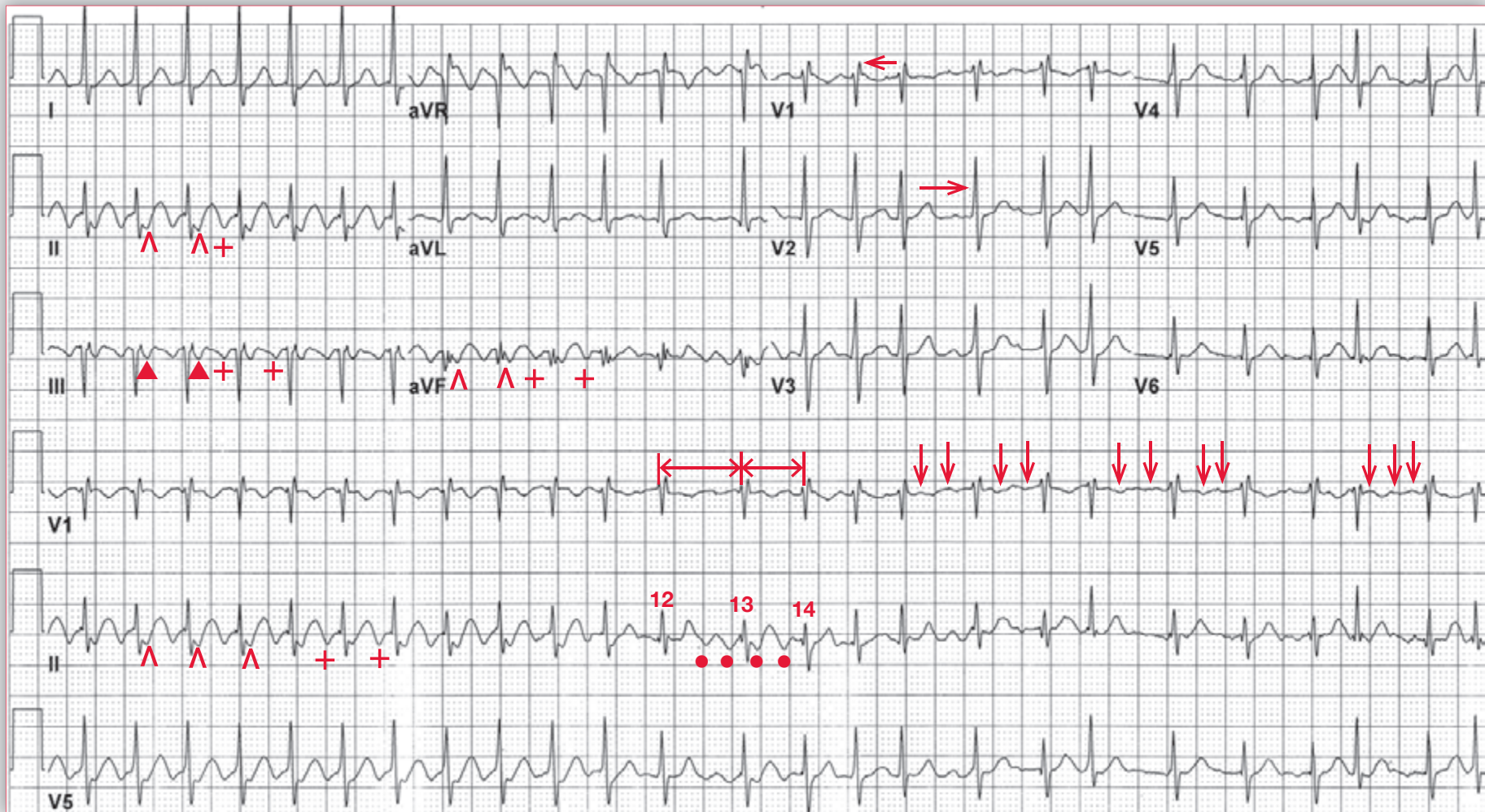
Although there are many causes for atrial fibrillation, one of the most common is hypertension, especially when left ventricular hypertrophy is present. The first step in treatment is rate control, especially when the patient is symptomatic as a result of a rapid rate. This involves the use of an AV nodal blocking agent, *ie*, β -blocker, calcium-channel blocker (verapamil or diltiazem), or digoxin. Electrocardioversion is appropriate if there is hemodynamic impairment. ■

Notes

A 38-year-old man is referred to an electrophysiologist for consideration of atrial flutter ablation. At the consultation visit, his prior ECGs are reviewed and the following ECG is obtained.

What does it show and what are the implications for his treatment?





ECG 57 Analysis: Atrial flutter (typical) reverting to atrial fibrillation, counterclockwise rotation, right ventricular conduction delay

ECG 57 shows the first 11 QRS complexes are regular at a rate of 160 bpm. The QRS complex duration is normal (0.08 sec), and there is a small R' (←) seen in lead V1, which is a minimal right ventricular conduction delay. There is a normal axis normal between 0 and +90 (positive QRS complex in leads I and aVF). Also noted is counterclockwise rotation with a tall R wave in lead V2 (→). This is determined by imagining the heart as viewed from under the diaphragm. With counterclockwise rotation, the left ventricular forces are shifted anteriorly and are seen in the early precordial leads, *ie*, V2. The QT/QTc intervals are normal (280/430 msec).

There is atrial activity (+), best seen in leads II, III, and aVF. The waveform is negative. Although no other atrial activity is clearly obvious, there is a suggestion of a second atrial waveform (▲) in the early part of the ST segment, most obvious in lead III. In lead II and aVF this waveform looks like ST-segment depression (^). In addition, between QRS complexes 12–13 and 13–14, there are long RR intervals (↔) and during this interval regular atrial waveforms can be seen at a rate of 320 (●). There is no isoelectric baseline between these waveforms. By using this PP interval, it can be seen that the second waveform seen in lead III (▲) is indeed an atrial wave. Also, what appears to be ST-segment depression in leads II and aVF (^) is actually the second atrial waveform. Hence the arrhythmia is typical atrial flutter with 2:1 AV block.

While the first 11 QRS complexes occur at a regular interval, the remaining QRS complexes, which have the same duration, morphology, and axis are irregularly irregular (average rate 140 bpm). The regular atrial flutter waves are no longer present. In contrast, there is a relatively flat baseline between each QRS complex) with low amplitude undulations (↓). Importantly, there is no apparent ST-segment depression in lead II, confirming the fact that this was indeed a flutter wave and not actually ST-segment depression as a result of ischemia. The rhythm is now atrial fibrillation. Hence in this patient atrial flutter with 2:1 AV block reverted to atrial fibrillation with a rapid response rate.

It is common for atrial flutter to convert to atrial fibrillation. While atrial flutter is a single reentrant circuit located in the right atrium, atrial fibrillation is the result of multiple reentrant circuits involving both the right and left atria. Both atrial arrhythmias can be treated initially with rate control although atrial flutter is often a more difficult rhythm to rate control. Definitive therapy involves reversion and prevention of the arrhythmia. Although atrial fibrillation may often be a permanent rhythm for patients once rate control is achieved, chronic atrial flutter may be problematic because adequate and consistent rate control may be difficult. Hence atrial flutter should be reverted and

continues

Notes

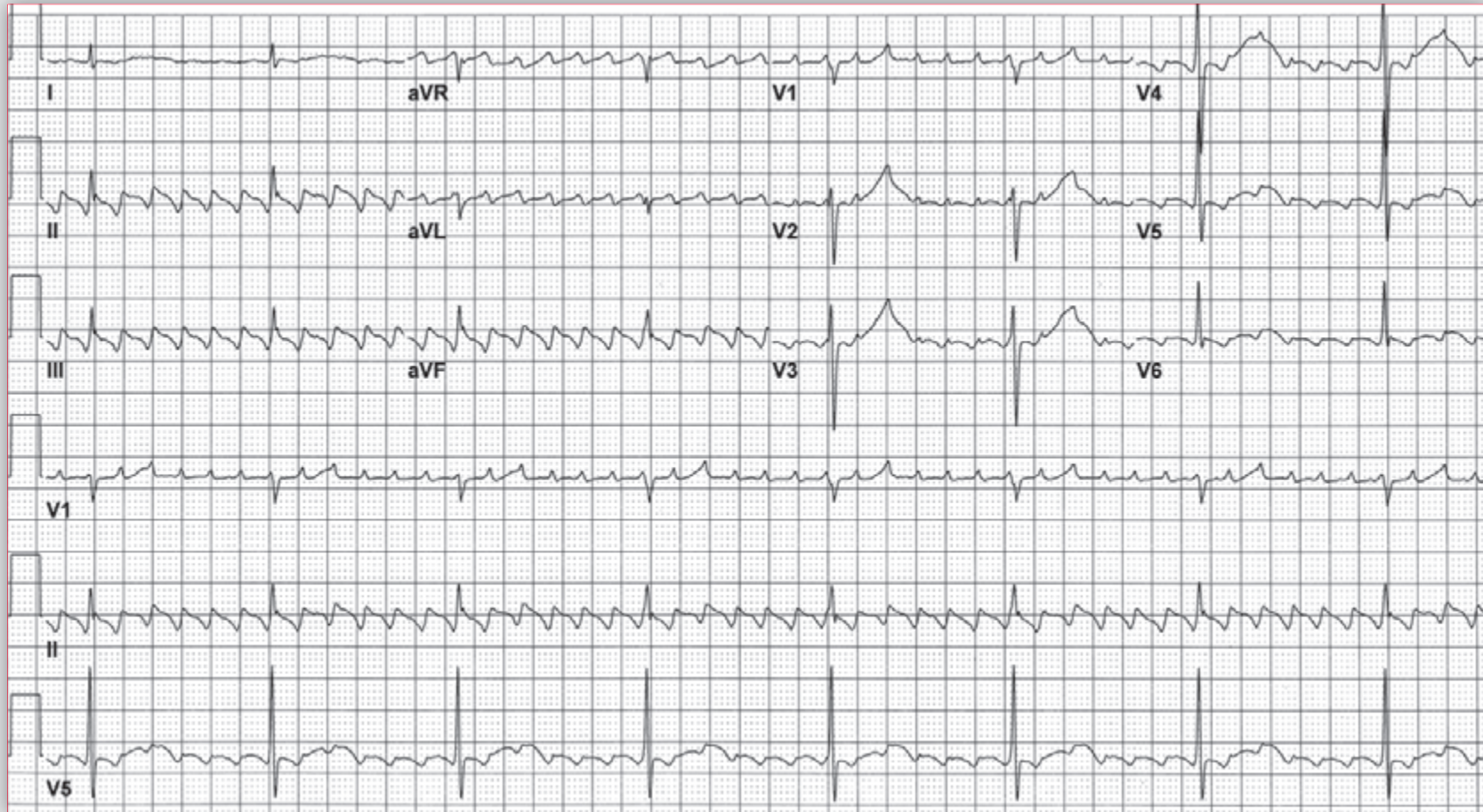
prevented. These atrial arrhythmias can be cardioverted with electrocardioversion or they may revert with an antiarrhythmic agent (class IA, IC or III). An antiarrhythmic agent is often prescribed for prevention of a recurrence once the arrhythmia is reverted. Another non-pharmacologic approach is radiofrequency ablation. Typical atrial flutter is “isthmus dependent” as a result of an area of slow conduction in the isthmus area between the tricuspid annulus and the inferior vena cava. As a result radiofrequency ablation of this area is a very effective

approach to therapy and is often curative. However, ablation for atrial fibrillation is more difficult, as it involves entering the left atrium transeptally and then delivering a line of ablation around the pulmonary veins (pulmonary vein isolation) as well as other ablation lines in other parts of the atrium. Although short-term results are reasonable, long-term results are not as good and hence this procedure is a treatment and not a cure. Currently it is used for patients with symptomatic AF that does not respond to pharmacologic therapy. ■

Core Case 58

A 60-year-old man is admitted with a tachyarrhythmia, and an ECG is obtained (ECG 58A). His rate is controlled with escalating doses of β -blockers. On the second day, ECG 58B is obtained.

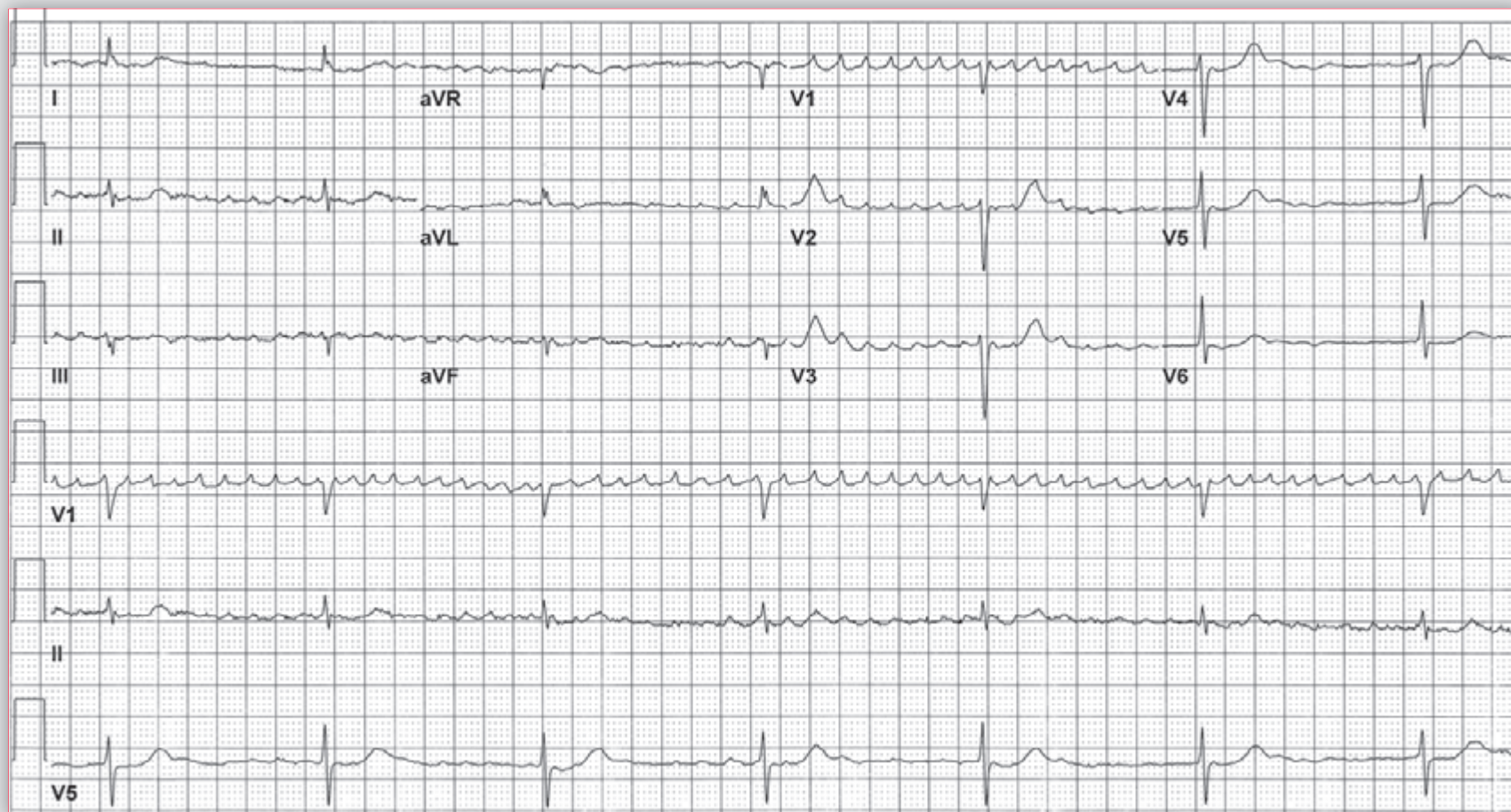
ECG 58A

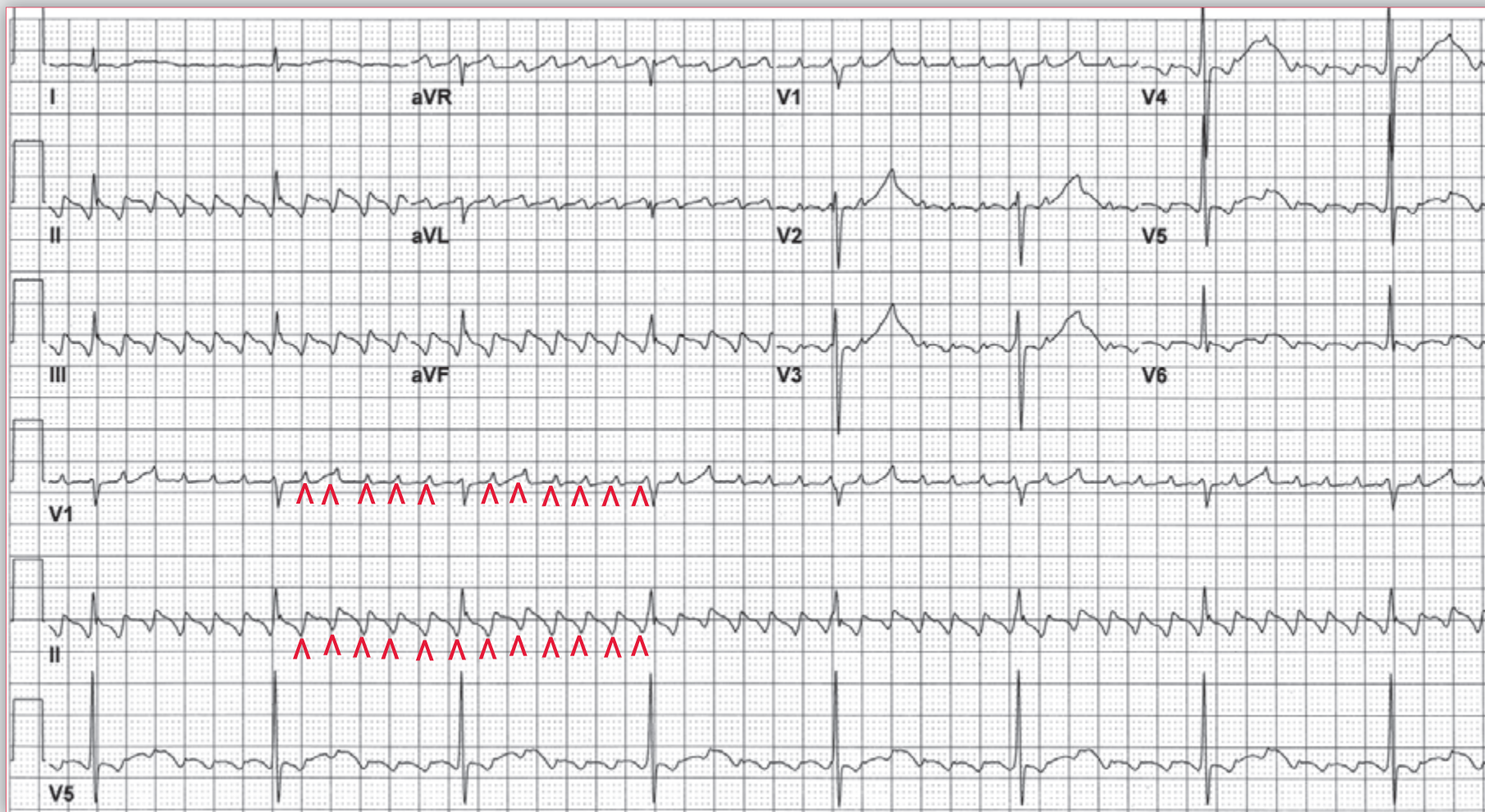


What do the ECGs show?

What is the likely mechanism of this phenomenon?

ECG 58B





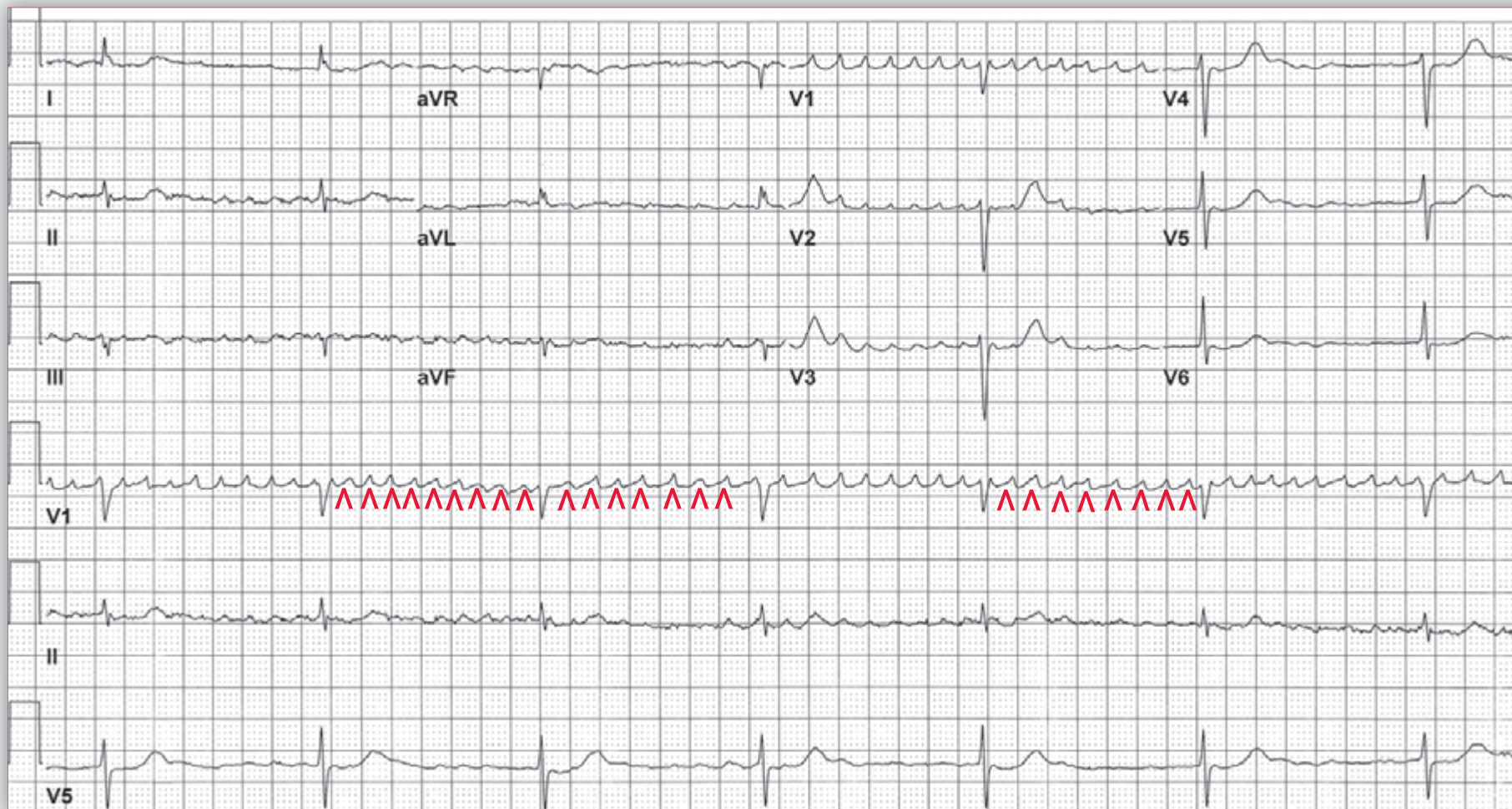
ECG 58A Analysis: Atrial flutter with 6:1 AV block, low voltage in limb leads

ECG 58A shows a regular rhythm at a rate of 48 bpm. The QRS complex have a normal duration (0.08 sec) and axis between 0° and $+90^{\circ}$ (positive QRS complex in leads I and aVF). There is low voltage in the limb leads (< 5 mm in each limb lead). The QT/QTc intervals are normal (480/430 msec). In between each QRS complex, there are prominent atrial waveforms (^) at a rate of 300 bpm. These atrial waveforms are regular in amplitude, morphology and interval. They are biphasic

(*ie*, negative–positive) in leads II, III, and aVF, and there is no isoelectric baseline between each atrial waveform. This is, therefore, typical (counterclockwise) atrial flutter with 6:1 AV block. It is not certain if the regular RR intervals at this slow rate represent a high degree of AV nodal conduction slowing or actually complete heart block with an escape junctional rhythm.

continues

Podrid's Real-World ECGs



ECG 58B Analysis: Coarse atrial fibrillation, regularization of RR intervals due to complete (3rd degree) AV block with junctional escape rhythm

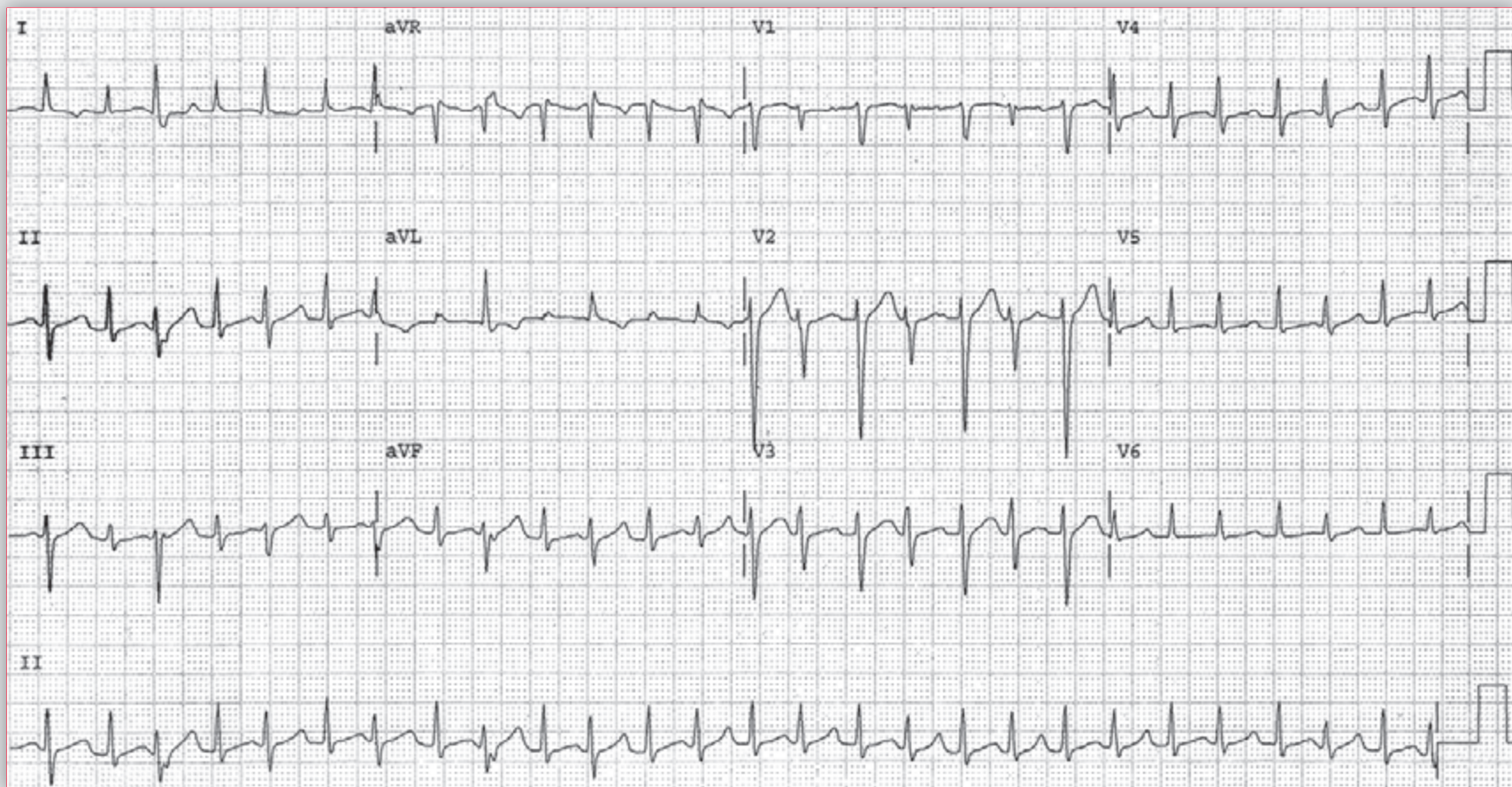
ECG 58B is from the same patient as ECG 58A. There is a regular rhythm at a rate of 42 bpm. The QRS complex duration and morphology and the QT/QTc intervals are identical to that seen in ECG 58A. However, the axis is now leftward between 0° and -30° (positive QRS complex in leads I and II and negative in lead aVF). The QT/QTc intervals are the same as in ECG 58A. There are no organized P waves seen. Between each QRS complex, there is atrial activity (^) that is coarse and looks like atrial flutter waves. However, the waveforms are irregular in amplitude, duration, and morphology. Therefore, this is not atrial flutter (as was seen in ECG 58A). In atrial flutter, the waveforms are completely regular in amplitude, morphology and interval. In contrast, these atrial waveforms are irregular; hence this is coarse atrial fibrillation. Although this has commonly been called “fib-flutter,” it does not actually exist normally as the atria may be either in atrial fibrillation or atrial flutter, but not both simultaneously. However, it is not uncommon for the rhythm to vary between atrial flutter and atrial fibrillation. With atrial fibrillation the RR intervals are irregularly irregular, while the RR intervals in atrial flutter are regular, or if irregular, they have a pattern based on the degree of AV block (hence

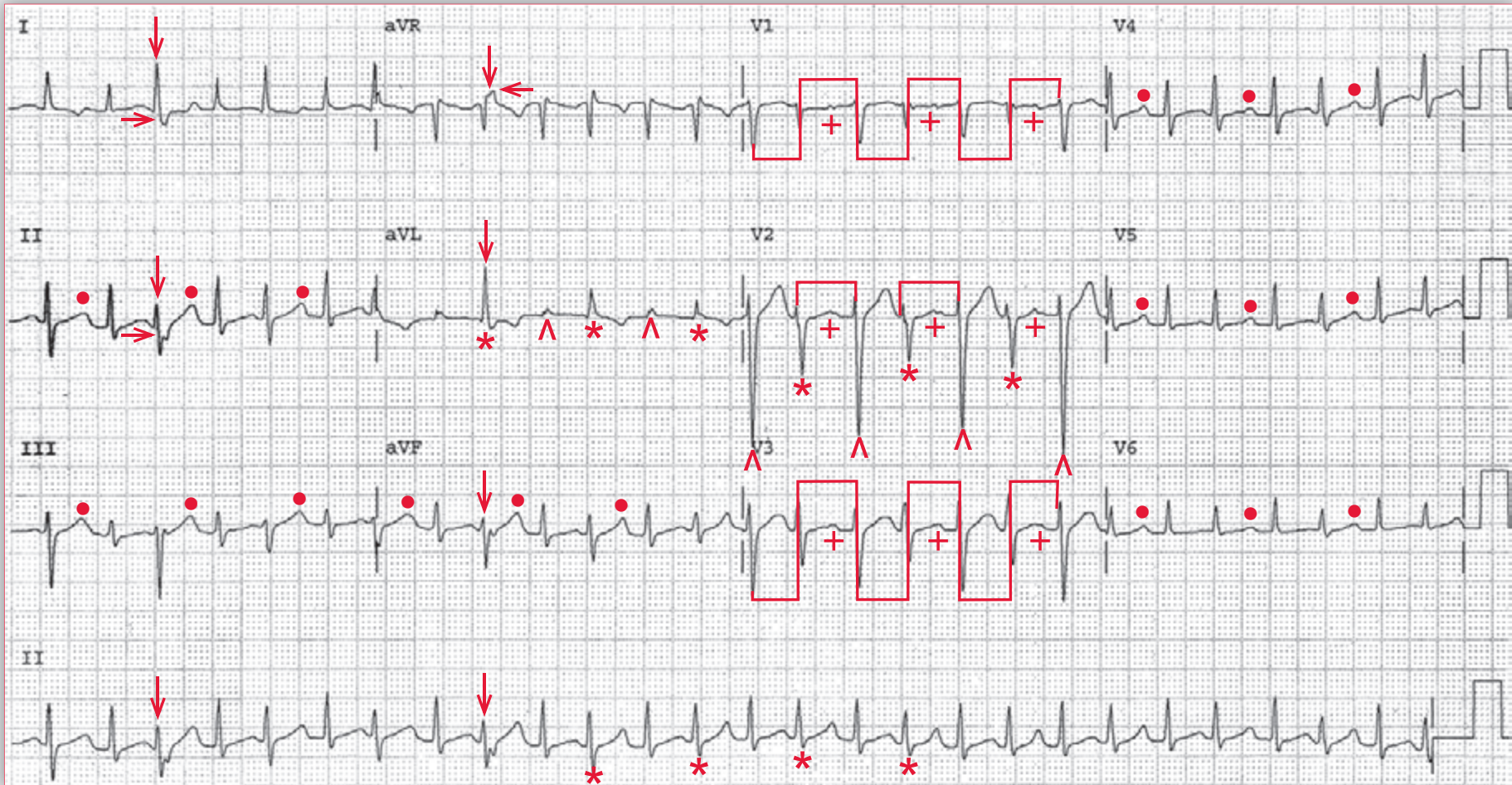
termed regularly irregular). In this ECG, the rhythm is atrial fibrillation, but the RR intervals are regular. Hence there is complete heart block present. The escape rhythm has QRS complexes that have the same duration and morphology as seen in ECG 58A. Hence this is an escape junctional rhythm. The change in the axis from normal, seen when the QRS complex results from conduction through the normal AV node, to a left axis when there is a junctional complex, is commonly seen. The junctional complex results from an ectopic focus, which can enter the bundle of His, which is a series of parallel pathways, at a slightly different location compared to the impulse that comes through the AV node. Hence a junctional QRS complex may have a different axis and/or a different amplitude when compared to a QRS complex originating above the AV node and conducted through the AV node. The fact that this is an escape junctional rhythm that has a shift in axis means that the QRS complexes noted in ECG 58A are not junctional, but are the result of conduction through the AV node. Hence the flutter is associated with a high degree of AV conduction slowing, but not complete heart block. ■

Notes

A 32-year-old man is observed to have an irregular heart rate on telemetry during an admission for alcohol withdrawal.

What does the ECG show?





ECG 59 Analysis: Sinus rhythm with frequent interpolated premature junctional complexes in a bigeminal pattern (junctional bigeminy), occasional rate-related right bundle branch block

ECG 59 shows that although the rhythm appears to be a regular tachycardia at a rate of 168 bpm, it can be seen that the rhythm is actually irregular and there is a regular pattern of longer (□) (0.42 sec) and shorter (◻) (0.36 sec) RR intervals. Therefore, every other QRS complex is premature (*). Each of the QRS complexes have a normal duration (0.08 sec), but the premature QRS complexes (*) have a different amplitude and a slightly different axis and slightly different T-wave amplitude (most apparent in leads V2–V3) compared to the other QRS complexes (^). While this resembles electrical or QRS alternans, this is not a regular rhythm, as every other QRS complex is premature. Electrical alternans is diagnosed when there is a regular RR interval. Although there are no P waves immediately obvious, close inspection of leads V1–V3 shows P waves (+) with a stable PR interval (0.16 sec) before the QRS complexes that are not premature, *ie*, those occurring after the longer RR interval (^). Correlating with the timing of these P waves are the more prominent positive T waves seen in leads II, III, aVF, and V4–V6 (●). These T waves have a greater amplitude than the T waves of the premature complexes and are the result of a P wave superimposed on the T wave. The P waves occur at a regular interval, and they are positive in leads II, aVF, and V4–V6. Thus, there is a regular sinus rhythm at a rate of 86 bpm.

Every other QRS complex is occurring after a shorter RR interval and hence is premature (*). The QRS complex duration and morphology

are identical to the sinus QRS complexes, although in some leads they have different amplitudes. However, there is no P wave before the premature QRS complex. Hence these are premature junctional complexes, occurring in a bigeminal pattern. The premature junctional complexes are termed interpolated as there is no pause after these complexes and they are not altering the PP interval surrounding them. It is very common for junctional complexes to have a different amplitude and/or axis compared to the sinus complexes, as the ectopic junctional focus originates from a different part of the junction compared to where the AV node (which conducts the sinus P wave) is located. The impulse from the ectopic focus enters the His-Purkinje system (which is a series of parallel pathways) at a slightly different location compared to the sinus complex, resulting in a slight change in QRS amplitude and/or axis.

It should also be noted that several of the premature junctional complexes (↓) are wide (0.14 sec) and have a right bundle branch block morphology with a broad S wave in leads I and II (→) and R' in lead aVR (←). This represents a rate-related right bundle branch block.

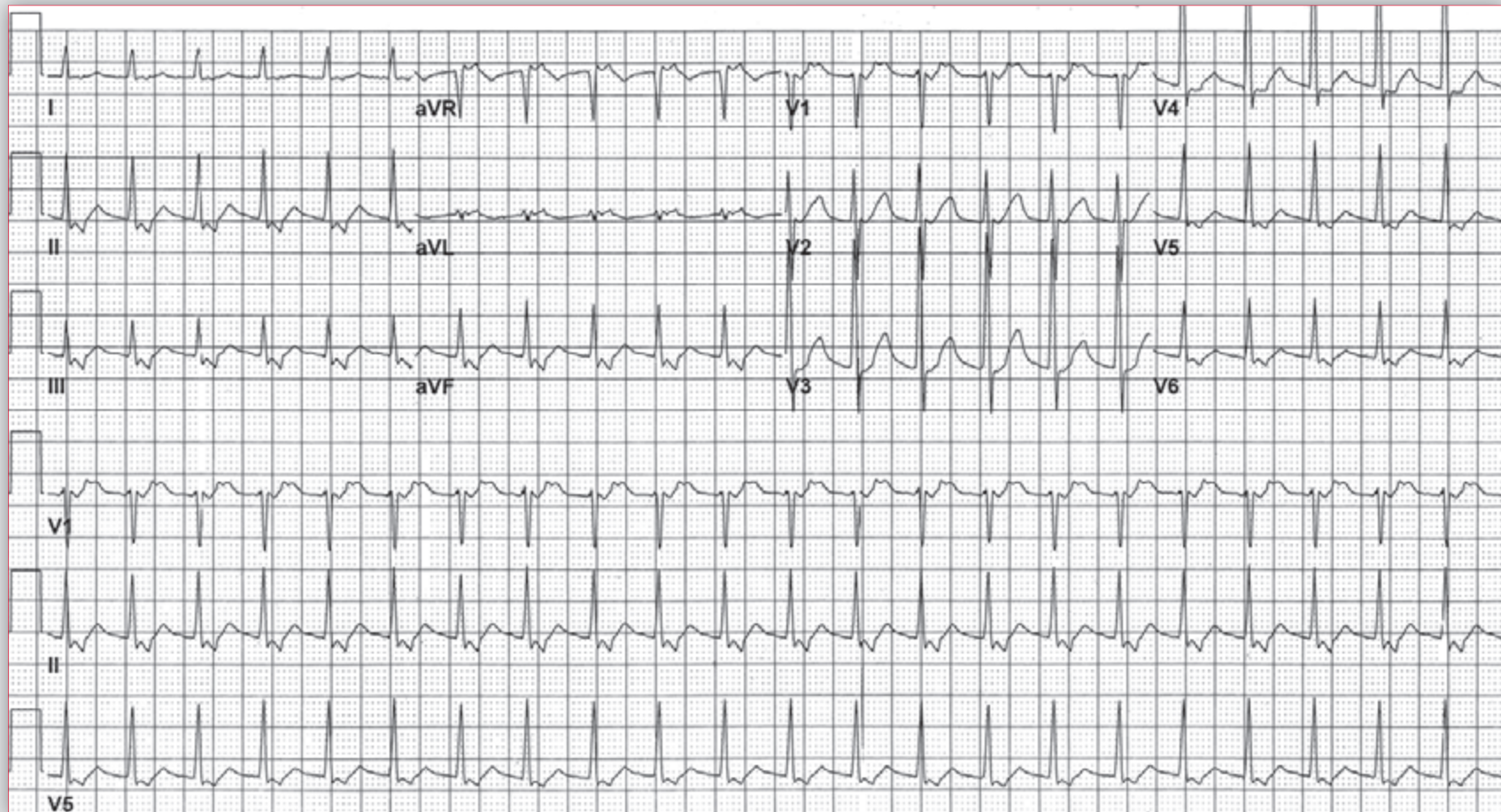
Therefore, while the average rate is 168 bpm, the actual sinus rate is 86 bpm. This is not truly a tachyarrhythmia as the rapid rate is a result of the premature junctional complexes. The QT/QTc intervals are slightly prolonged (280/460 msec) but this is not accurate as it is not clear what underlying rate should be used to correct the QT interval. ■

Core Case 60

A 56-year-old man without any cardiac history presents to an emergency department with complaints of palpitations that he noted after completing a 3-mile jog. He has no previous cardiac history, although he has been told of hypertension in the past. He has not seen a physician for several

years and has not taken any medications. He had no symptoms while jogging, but after finishing, he noted that his heart rate failed to slow and he was now aware of palpitations. He is initially seen by a resident in the emergency department who notes that his physical examination is unremarkable

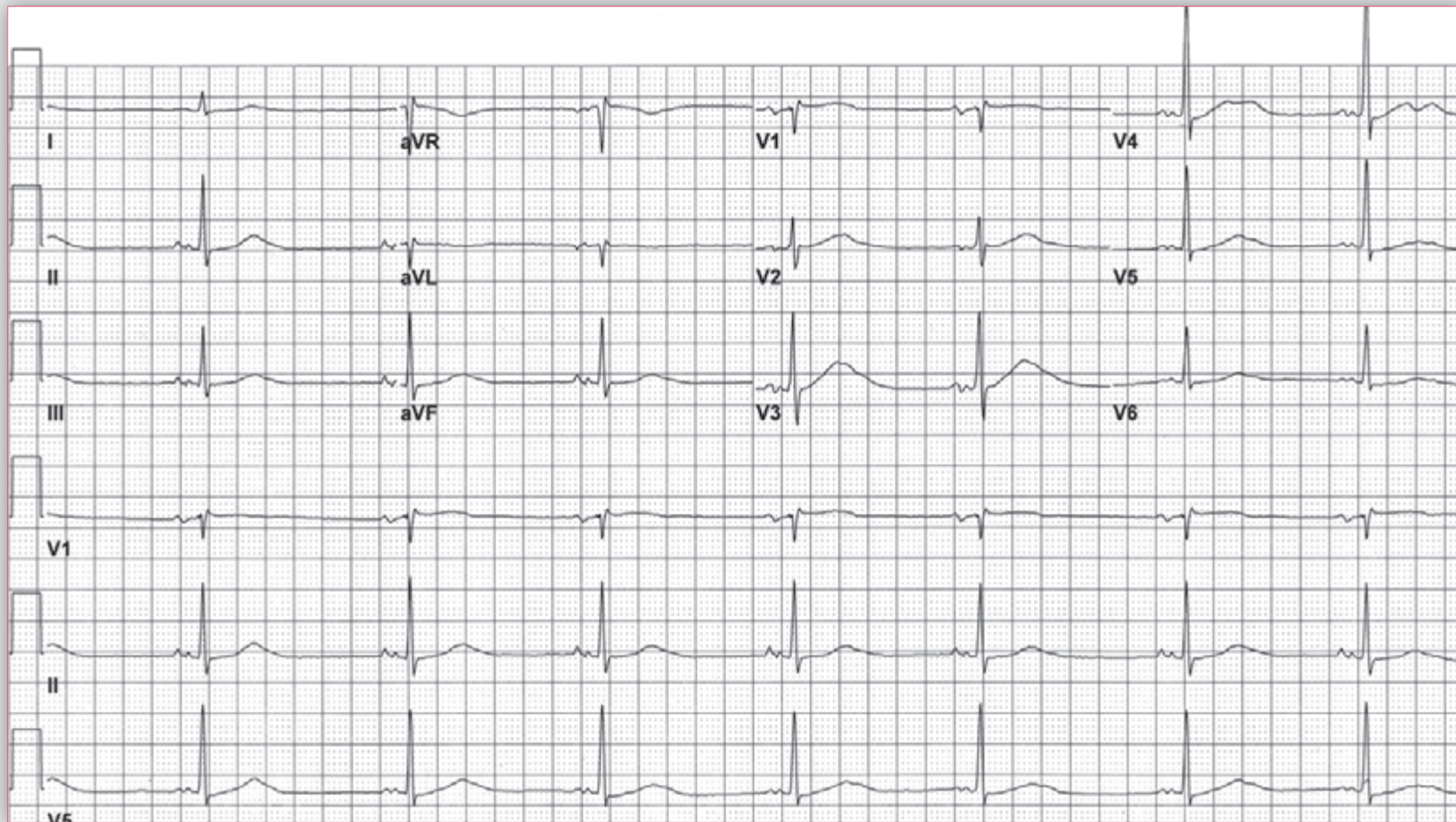
ECG 60A



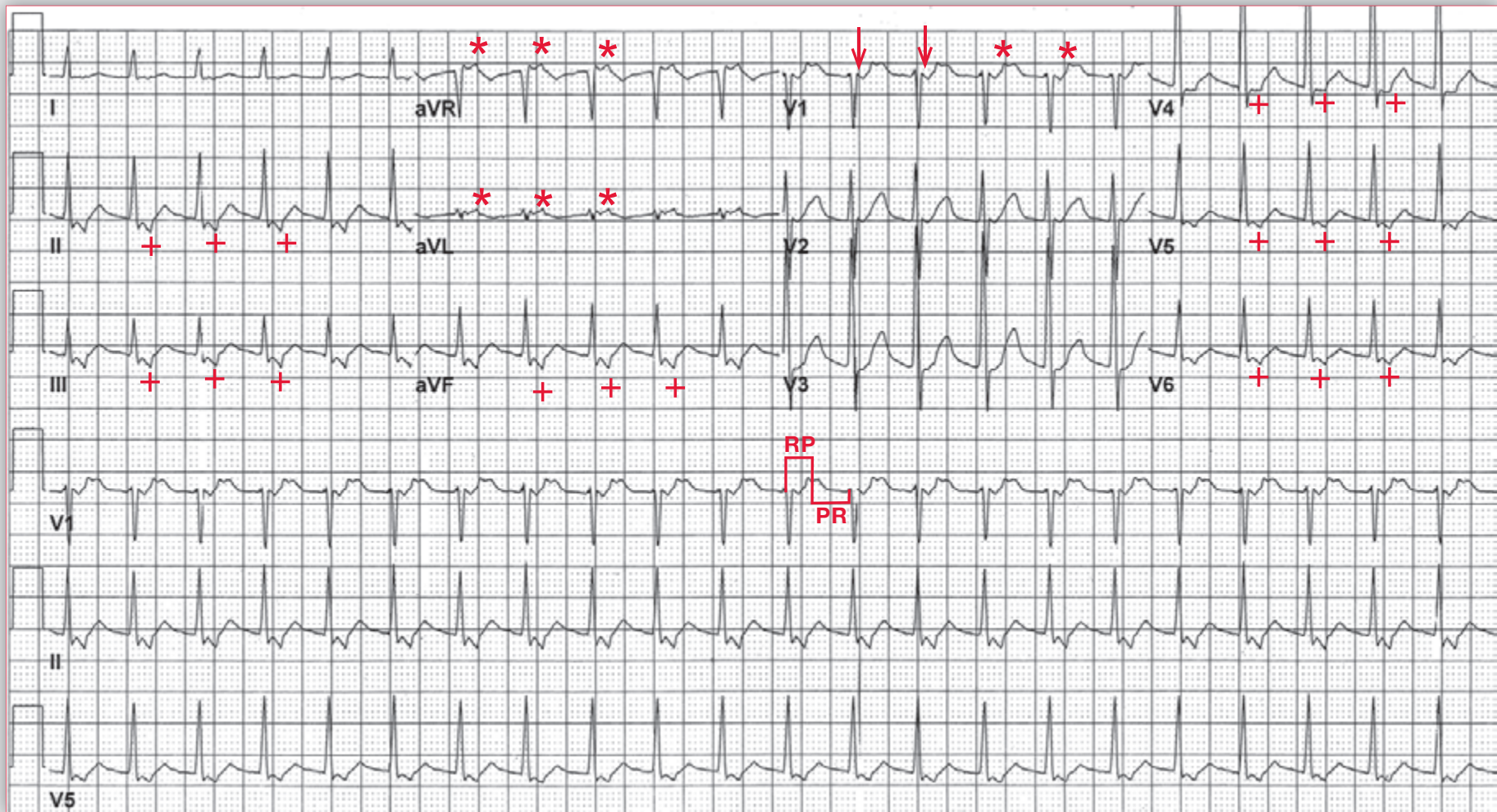
except for a tachycardia. An ECG is obtained (ECG 60A). After the attending arrives and examines the patient, he performs carotid sinus pressure, and the patient notes a sudden decrease in his heart rate and a resolution of the symptoms. A repeat ECG is obtained (ECG 60B).

What is the differential diagnosis for the underlying condition seen in the ECGs?

ECG 60B



Podrid's Real-World ECGs



ECG 60A Analysis: Narrow complex supraventricular tachycardia (short-RP tachycardia)

ECG 60A shows a regular rhythm at a rate of 134 bpm. The QRS complex duration is normal (0.08 sec) with a normal morphology, although there is a small R' in V1 (↓), which is a normal variant representing a slight right ventricular conduction delay, and a normal axis between 0° and +90° (positive QRS complex in leads I and aVF). The QT/QTc intervals are normal (290/430 msec). There are no P waves seen before any of the QRS complexes. However, there is a P wave seen after each QRS complex in leads V1, aVR, and aVL, where there is a positive deflection (*). Correlating with this waveform is a negative deflection within the ST segment, most prominently seen in leads II, III, aVF, and V4–V6 (+); this is a negative P wave that resembles ST-segment depression. The RP interval (0.14 sec) is shorter than the PR interval (0.28 sec). Hence this is a short-RP supraventricular tachycardia. Etiologies include sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atrioventricular reentrant tachycardia, and typical or common atrioventricular nodal reentrant tachycardia of an unusual type (slow-slow). As the P wave is negative in leads II, aVF, and V4–V6, this is not a sinus tachycardia. The absence of a second atrial waveform between each QRS complex eliminates atrial flutter with 2:1 AV block. Hence the etiologies include an atrial tachycardia, an ectopic junctional tachycardia, an uncommon form of typical AVNRT (slow-slow), or AVRT.

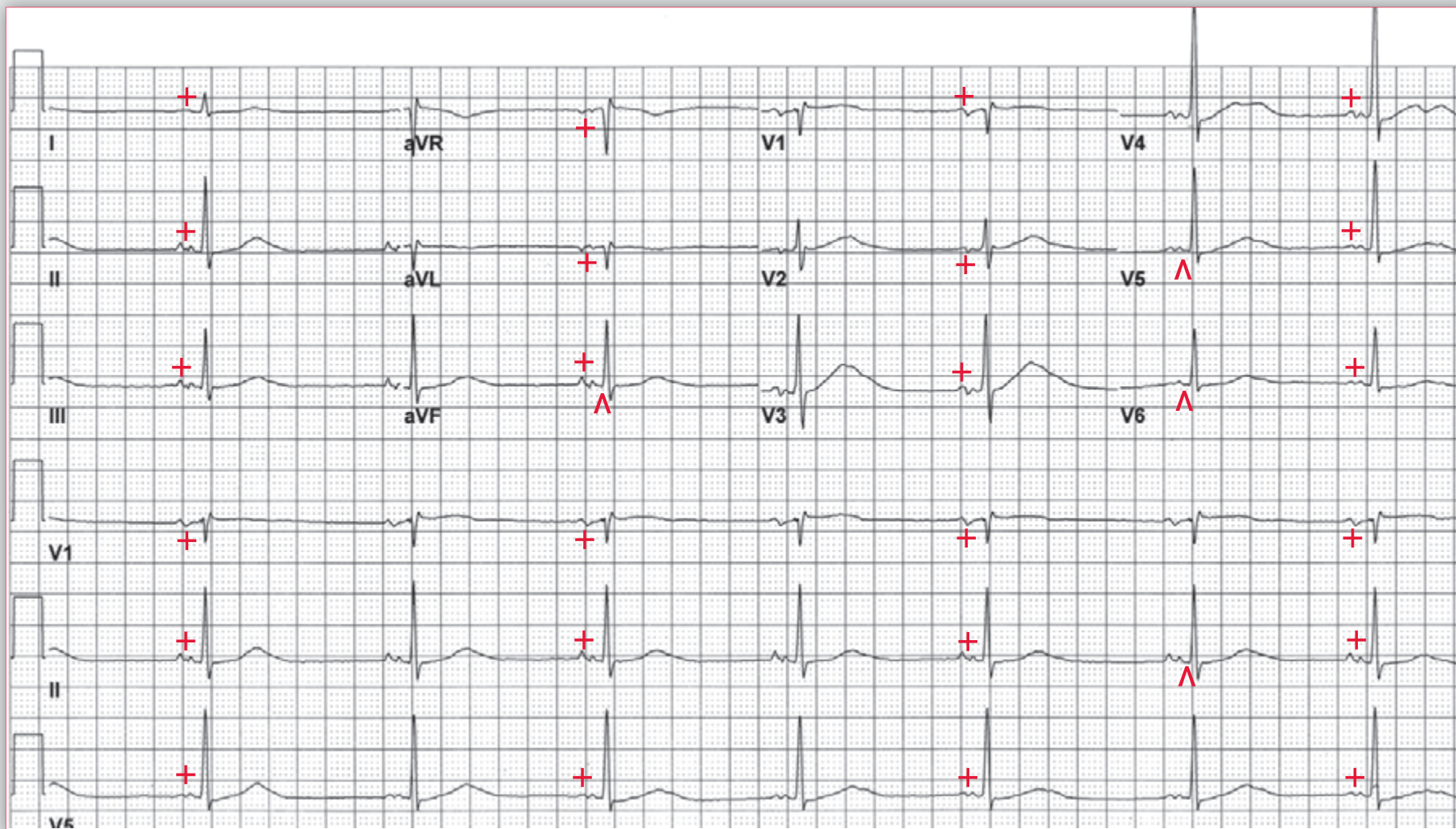
An AVRT is a supraventricular tachycardia associated with an accessory pathway and often evidence of a preexcitation pattern. The preexcitation pattern may be Wolff-Parkinson-White (WPW), in which the accessory pathway is an atrial-ventricular pathway that serves as a second pathway way from the atrium to ventricle. In this situation the

baseline ECG shows a short PR interval and widened QRS complex as a result of a delta wave, which is due to early ventricular myocardial activation by the accessory pathway that, by bypassing the normal AV node, results in early (preexcited) ventricular activation. The two distinct AV pathways (accessory pathway and normal AV node–His–Purkinje pathway) are linked in the atria and ventricles, forming a circuit around which the impulse can circulate, generating an AVRT. When the activation to the ventricle is via the normal AV node–His–Purkinje system, the AVRT is termed orthodromic and the QRS complex has a normal supraventricular morphology. If the activation to the ventricle is via the accessory pathway, the AVRT is termed antidromic and the QRS complex is wide and preexcited, resembling the complex seen in normal sinus rhythm. The Lown-Ganong Levine pattern is a short PR interval, as a result of a bypass tract that begins in the atrium and connects to the bundle of His. Hence the short PR interval is associated with a narrow or supraventricular QRS complex. There may also be an accessory pathway that links the atrium to the ventricle, but which only conducts retrogradely. This is termed a concealed bypass tract. In this situation, the baseline ECG is normal.

An AVNRT is due to a reentrant arrhythmia generated within the AV node as a result of a circuit involving dual AV nodal pathways, *ie*, a fast pathway that conducts rapidly but recovers slowly (long refractory period) and a slow pathway that conducts slowly but recovers more rapidly (short refractory period). A typical AVNRT involves antegrade conduction to the ventricles via the slow pathway and retrograde conduction back to the atria via the fast pathway (termed slow-fast). In

continues

Podrid's Real-World ECGs



ECG 60B Analysis: Sinus bradycardia, left atrial hypertrophy (left atrial abnormality), possible Lown-Ganong-Levine pattern

this situation, there is no P wave seen as there is simultaneous atrial and ventricular activation. An uncommon variant of this is termed slow-slow, *ie*, the fast pathway is relatively slow conduction (termed slow-slow), resulting in a slight delay in retrograde atrial activation and presenting as a short PR tachycardia.

An ectopic junctional tachycardia is due to an ectopic focus in the junction that initiates an impulse faster than the sinus node and hence becomes the dominant pacemaker. It is not based upon a reentrant circuit, but rather enhanced automaticity.

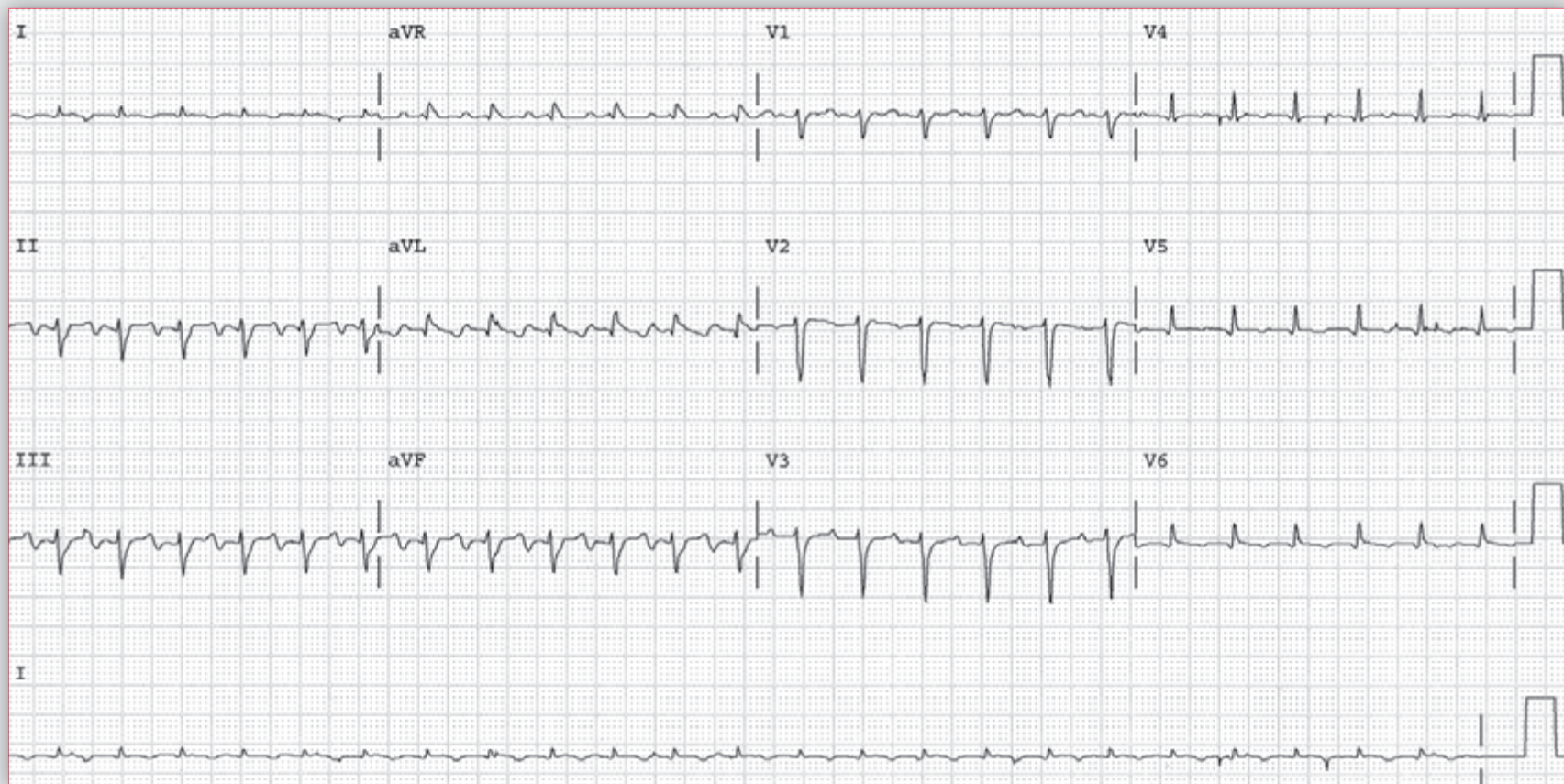
ECG 60B shows a regular rhythm at a rate of 44 bpm. The QRS complex is of normal duration, axis, and morphology; it is identical to the QRS complex seen in ECG 60A. The QT interval is prolonged (500 msec) but the QTc interval is the same (430 msec) as in ECG 60A. There is a P wave (+) before each QRS complex with a stable PR interval (0.18 sec). The P wave is positive in leads I, II, aVF, and V4–V6; hence this is a sinus bradycardia. The P-wave morphology is abnormal, and it is broad and prominently notched in leads II, III, aVF,

and V3–V5. This is either an intraatrial conduction delay or left atrial hypertrophy (often called a P mitrale). Although the PR interval is normal, it can be seen that there is a very short or non-existent PR segment. As the PR interval includes the P wave and PR segment, a broad P wave may result in a normal PR interval. However, the PR segment correlates with AV conduction through the AV node and His-Purkinje system. In this case the short or non-existent PR segment in several leads (^) suggests an accessory pathway bypassing the AV node. As the QRS complex is normal in duration, this probably indicates that there is a Lown-Ganong-Levine pattern. The inverted waveforms altering the ST segment, seen in ECG 60A, are no longer present. This confirms the fact that these waveforms were P waves and that the rhythm in ECG 60A was a short-RP tachycardia. The etiology is still not established based on the ECG; however, the response to carotid sinus pressure, with termination of the arrhythmia, strongly suggests that the mechanism was reentry and that the AV node was part of the circuit. Hence the etiologies would be either an AVNRT or an AVRT. The short PR segment and possible Lown-Ganong-Levine pattern suggests that the rhythm is AVRT. ■

Core Case 61

A 60-year-old man is seen for complaints of palpitations that began two hours before being seen. He does not have any other complaints and has never had palpitations in the past. He does relate that he has been under

ECG 61A

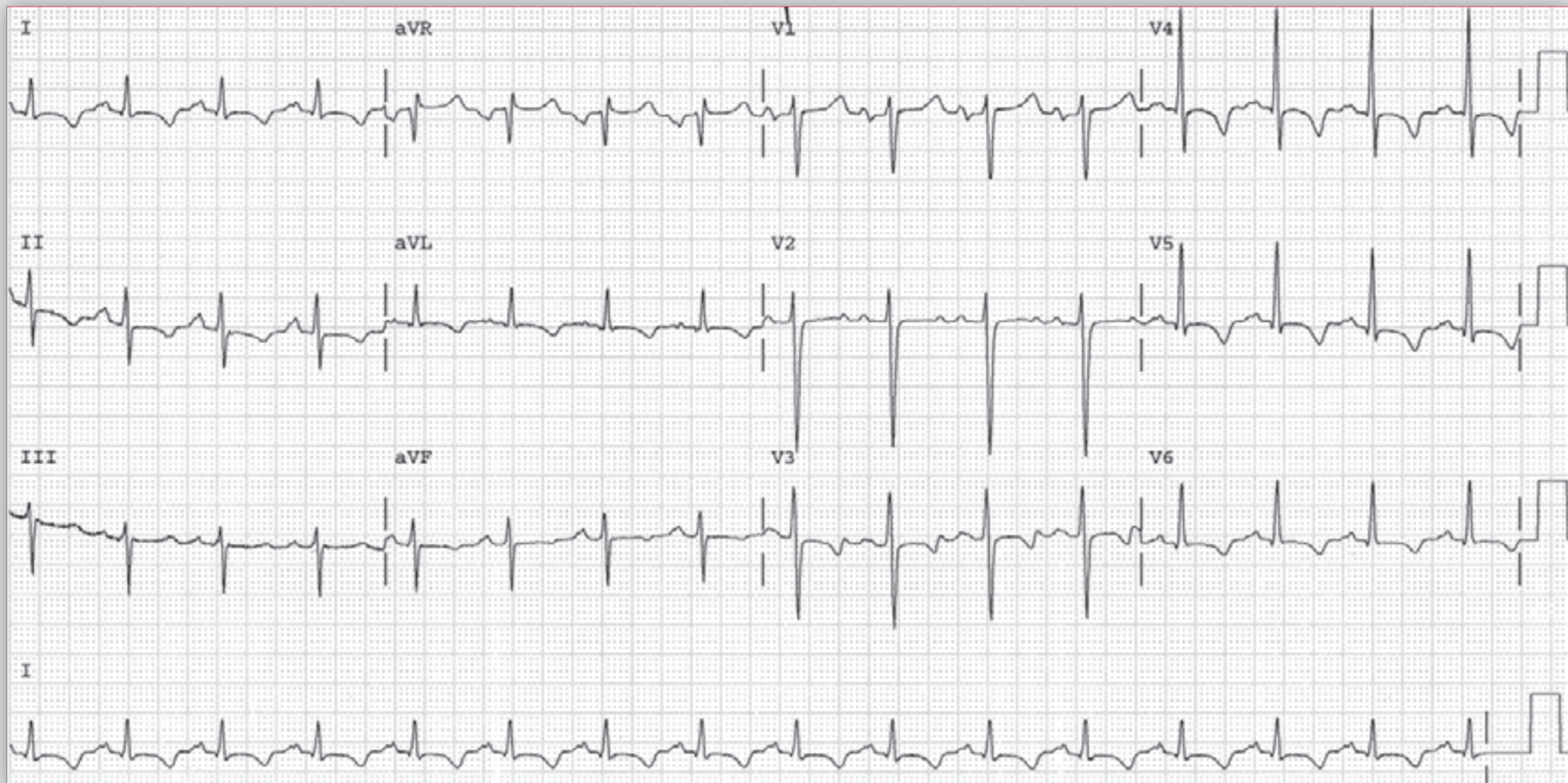


more stress at home and drank more coffee than usual prior to the onset of his palpitations. An ECG is obtained (ECG 61A) and this is compared to his baseline ECG (ECG 61B) obtained 6 months prior to this visit.

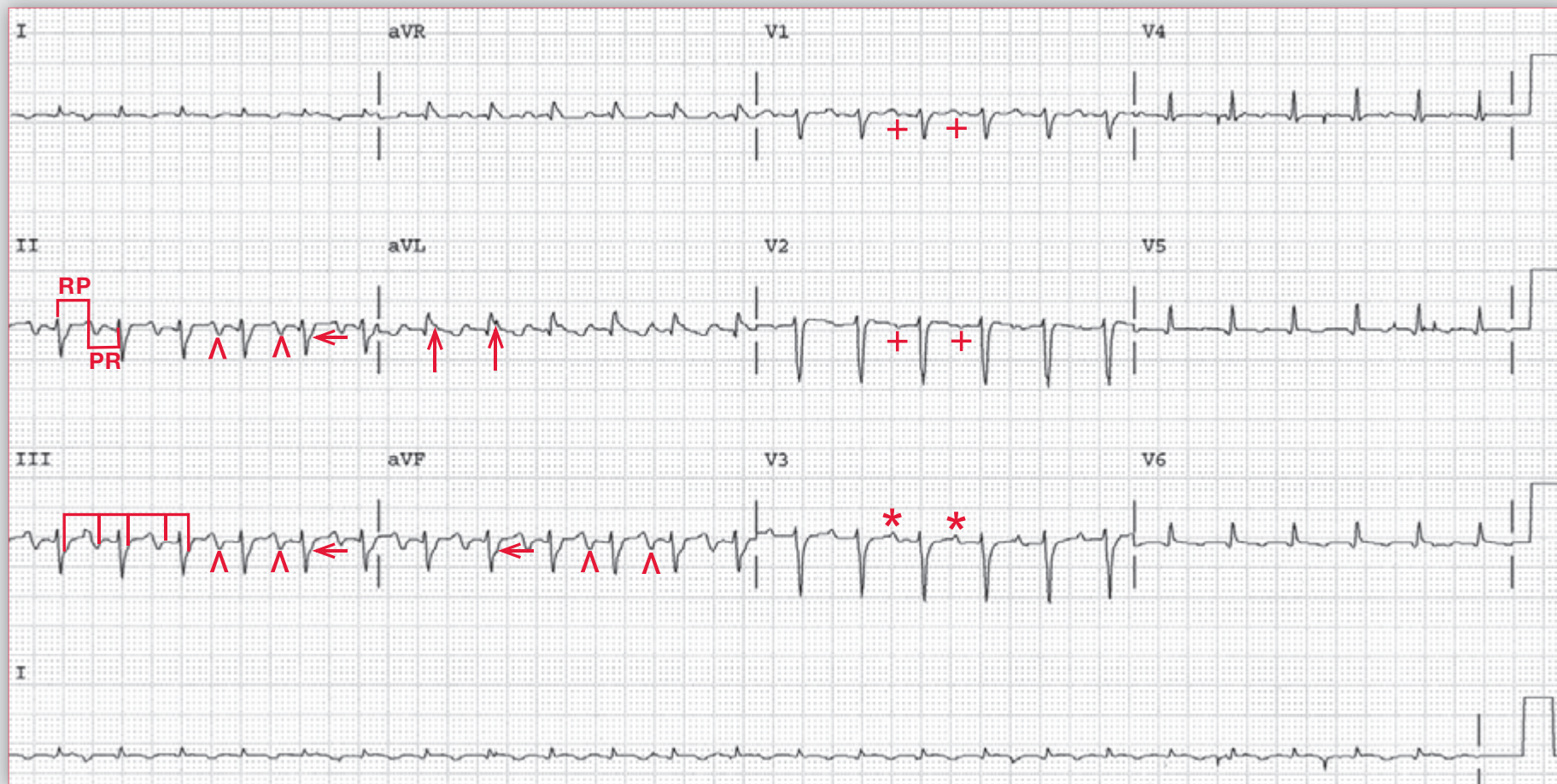
What do the ECGs show?

What is the underlying diagnosis in ECG 61A?

ECG 61B



Podrid's Real-World ECGs



ECG 61A Analysis: Narrow complex supraventricular tachycardia (long-RP tachycardia), atrial flutter with 2:1 AV block, low-voltage, left anterior fascicular block

ECG 61A shows a regular rhythm at a rate of 146 bpm. The QRS complex duration is normal (0.10 sec). There is low voltage throughout (< 5 mm in each limb lead and < 10 mm in each precordial lead). The axis is extremely leftward between -30° and -90° (positive QRS complex in lead I, and negative in leads II and aVF not due to an initial Q wave). This is a left anterior fascicular block. The QT/QTc intervals cannot be established, as there is no clear T wave seen in any lead.

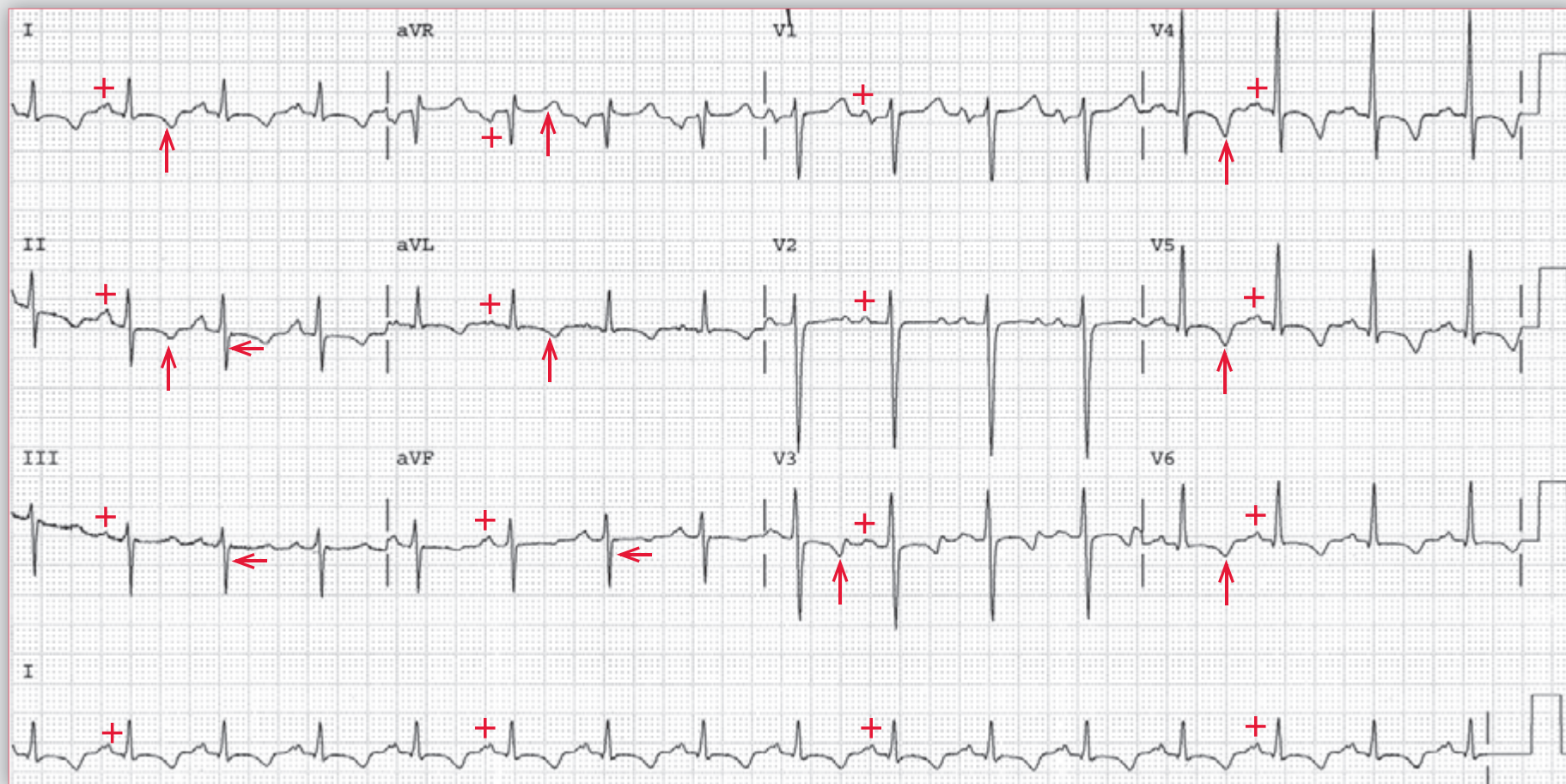
Although there are no obvious P waves seen, there are waveforms present in leads V1–V2 (+) that are atrial in origin. These correspond to a positive waveform (*) in lead V3 as well as negative waveforms (^) in leads II, III, and aVF that look like negative T waves. However, these are negative atrial waveforms. This is termed a long-RP tachycardia

(RP interval = 0.24 sec, PR interval = 0.18 sec). Etiologies for a long-RP tachycardia include: sinus tachycardia, atrial tachycardia, atrial flutter with 2:1 AV block, ectopic junctional tachycardia, atrioventricular reentrant tachycardia, and uncommon or atypical atrioventricular nodal reentrant tachycardia (fast-slow).

Less obvious is a second atrial waveform that can be seen at the end of the QRS complex in lead aVL (\uparrow). This corresponds to the terminal notching (\leftarrow) of the QRS complex seen in leads II, III, and aVF. The two atrial waveforms occur at a constant interval (\square) at a rate of almost 300 bpm. Hence the rhythm is atrial flutter with 2:1 AV block. Atrial flutter is the only arrhythmia at a regular atrial rate > 260 bpm.

continues

Podrid's Real-World ECGs



ECG 61B Analysis: Normal sinus rhythm, left axis, nonspecific ST-T wave changes

ECG 61B is from the same patient as ECG 61A. There is a regular rhythm at a rate of 96 bpm. The QRS complex amplitude is now normal and the axis is less leftward and is about -30° (QRS biphasic in lead II). There is a slight difference in the QRS duration, which is narrower (0.08 sec) than the QRS complex in ECG 61A. There is also a slight difference in QRS complex morphology with the absence of the terminal notching of the QRS complex in leads II, III, and aVF (\leftarrow). This supports the fact that the slight QRS widening and terminal notching was indeed the second flutter wave.

There is a P wave (+) before each QRS complex with a constant PR interval (0.20 sec). Hence this is normal sinus rhythm. Also noted are diffuse T-wave inversions (\uparrow) (positive T wave in lead aVR is actually inversion). The QT/QTc intervals are normal (360/450 msec).

Atrial flutter is often a difficult arrhythmia to diagnose as one of the two flutter waves may not be obvious, being at the end of the QRS complex and resembling ST-segment depression or an S wave, or at the beginning of the QRS complex resembling a Q wave. The normal atrial rate for atrial flutter is 260 to 320 bpm (most frequently 300 bpm) and most commonly there is 2:1 AV block, with a ventricular rate of 130 to 160 bpm (most frequently 150 bpm). Whenever there is a rapid supraventricular tachycardia at this rate, blockade of the AV node will result in slowing of AV nodal conduction and reduce the ventricular response rate, exposing the atrial waveforms. Based on the atrial rate as well as the atrial waveform morphology (in atrial flutter negative positive

waveforms without an isoelectric baseline between each waveform) atrial flutter can be established as the etiology. A second arrhythmia often confused with atrial flutter is atrial tachycardia. This arrhythmia has distinct P waves with an isoelectric baseline between them.

There are many causes for atrial arrhythmias, including atrial flutter. While often no definite precipitating factor can be identified, in this patient it is likely that the family stress coupled with an excessive intake of caffeine resulted in the first and only episode of atrial flutter. Initial treatment is aimed at rate control, which involved the use of an AV nodal blocking agent. Although it is possible that the atrial flutter will revert spontaneously to NSR after the levels of caffeine decrease, therapy for continued atrial flutter would be either pharmacologic cardioversion (as with IV ibutilide, for example) or electrocardioversion. As the palpitations clearly began two hours before being seen, he could be safely cardioverted without prior anticoagulation. Current approach supports cardioversion without anticoagulation for atrial flutter or atrial fibrillation that is of < 48 hours duration. For arrhythmia of > 48 hours or unknown duration, anticoagulation for 3 to 4 weeks before cardioversion is recommended. If a patient with arrhythmia of > 48 hours duration is hemodynamically unstable or very symptomatic despite adequate rate control, cardioversion without anticoagulation can be performed after a transesophageal echocardiogram is performed, confirming the absence of thrombus in the left atrial appendage. However, anticoagulation for at least 4 weeks would be indicated after cardioversion. ■

Notes

Index

A

accelerated idioventricular tachycardia, 127
accelerated ventricular rhythm, 125
acute myocardial infarction, 127
anteroapical myocardial infarction, 124–127
anterolateral myocardial infarction, 124–127
anteroseptal myocardial infarction, 218–219, 284–287
antiarrhythmic drugs, 170–173
antidromic atrioventricular reentrant tachycardia, 144–145, 147, 163
Ashman phenomenon, 184–185, 188–191
atrial activated ventricular pacing, 180–181
atrial bigeminy, 2, 130–131
atrial fibrillation, 16–17, 141, 150–151, 153, 184–185, 316–317, 322–323, 334–335
 atrial flutter conversion to, 326–327, 329
 with rapid ventricular response, 62–63
atrial flutter, 56–57, 59, 141, 180–181, 351
 conversion to atrial fibrillation, 326–327, 329
 with 1:1 atrioventricular conduction, 276–277
 with 6:1 atrioventricular block, 332–333
 with 3:2 Wenckebach, 280–281
 with 2:1 atrioventricular block, 278–279, 348–349
 with 2:1 atrioventricular conduction, 268–269
 with variable block, 94–95, 270–271
atrial tachycardia, 44–45, 47, 84–85, 234–235, 238–239, 241, 244–245, 250–251
 with 2:1 AV block, 52–53, 246–247, 262–263
 with 3:1 AV block, 264–265
 with 1:1 conduction, 50–51
 with Mobitz type I second-degree atrioventricular block, 254–255
 multifocal, 258–259
atrial trigeminy, 7
atrioventricular block
 atrial tachycardia with, 52–53
 first-degree, 120–121, 168–173, 214–215
 5:4 Wenckebach, 254–255
 Mobitz type I, 246–247, 254–255, 300–301
 second-degree, 246–247, 254–255, 300–301
 6:1, 332–333

 third-degree, 334–335
 3:1, 264–265
 2:1, 52–53, 262–263, 348–349
 Wenckebach, 246–247, 254–255
atrioventricular conduction
 1:1, 56–57
 prolonged, 120–121, 168–173
atrioventricular dissociation, 112–113, 119, 124–125, 145
atrioventricular nodal reentrant tachycardia, 66–67, 72–73, 93, 101, 106–107, 188–191, 206–207, 209, 284–285, 292–295
 atrioventricular reentrant tachycardia versus, 300–301
 slow-slow, 51, 292–295, 313, 343, 345
atrioventricular reentrant tachycardia
 antidromic, 144–145, 147, 163
 atrioventricular nodal reentrant tachycardia versus, 300–301
 orthodromic, 162–163, 304–305, 307, 312–315
atrioventricular sequential pacing, 180–181
AV. *See* atrioventricular
AV node–His–Purkinje system, 135, 161, 163, 307, 319

B

Belhassen tachycardia, 113
bradycardia, sinus, 26–27, 108–109, 250–251, 300–301, 344–345
bundle branch blocks
 functional, 12
 left. *See* left bundle branch block
 right. *See* right bundle branch block

C

capture beat, 112–113
clockwise rotation, 234–235
concertina effect, 156–157
conduction delay, 68–69
 intraventricular, 20–21, 72–73, 75, 78–81, 120–121, 168–*173
 right ventricular, 262–263, 304–305, 326–327
counterclockwise rotation, 326–327
crista pattern, 269

D

diffuse ST-T wave abnormalities, 200–203
Dressler beat, 113
Dressler's syndrome, 281

E

early transition, 2–3
ectopic atrial rhythm, 240–241
electric alternans, 57, 189
electrocardiogram, 58–59, 68–69, 127
endless loop tachycardia, 177
escape junctional complexes, 26–27
escape ventricular rhythm, 125

F

fascicular block
 left anterior, 22–23, 66–67, 126–127, 202–203, 258–259, 348–349
 left posterior, 26–27, 50–51, 56–57, 120–121, 206–209, 222–225, 276–277, 279–281
fascicular tachycardia, 112–113
fast-slow atrioventricular nodal reentrant tachycardia, 93
first-degree atrioventricular block, 120–121, 168–173, 214–215

H

His–Purkinje system, 67, 79, 113, 189
hyperacute T waves, 202–203
hyperkalemia, 194–195, 197, 200–201, 203

I

infarction. *See* myocardial infarction
inferior myocardial infarction, 284–287
inferior wall myocardial infarction, 26–27
 old, 40–41, 50–53, 322–323
 pseudo, 134–135, 138–139, 306–307
intermittent rate-related left bundle branch block, 16–17
intermittent Wolff–Parkinson–White pattern, 134–135, 138–139, 160–161
interpolated premature junctional complexes, 338–339

intraventricular conduction delay, 20–21, 72–73, 75, 80–81, 120–121, 168–173
 sinus tachycardia with, 78–79
 isorhythmic dissociation, 124–125
 IVCD. *See* intraventricular conduction delay

J
 junctional bigeminy, 338–339
 junctional escape rhythm, 334–335
 junctional rhythm, 102–103, 202–203
 junctional tachycardia, 100–101

L
 LAFB. *See* left anterior fascicular block
 lateral myocardial infarction, 124–127, 258–259
 LBBB. *See* left bundle branch block
 left anterior fascicular block, 66–67, 126–127, 202–203, 258–259, 348–349
 rate-related, 22–23, 44–45
 left atrial hypertrophy, 12–13, 138–139, 160–161, 344–345
 left axis, 68–69, 228–229, 244–245, 350–351
 left bundle branch block, 14–15, 38–39, 106–107
 intermittent rate-related, 16–17
 premature atrial complex with, 12–13, 40–41
 rate-related, 6–7, 16–17, 34–35, 40–41, 162–163
 left bundle branch morphology, 108–109
 left posterior fascicular block, 26–27, 50–51, 56–57, 120–121, 206–209, 222–225, 276–277, 279–281
 left posterior fascicular tachycardia, 113, 278–279
 left ventricular hypertrophy, 40–41, 78–81, 322–323
 long PR tachycardia, 85
 long-RP tachycardia, 84–85, 92–93, 218, 222–223, 234–235, 238–239, 244–247, 250–251, 268–269, 304–305, 348–349
 low limb lead voltage, 74–75, 196–197, 218–219
 low QRS voltage, 228–229, 262–263
 low voltage, 50–51, 66–69, 84–87, 92–97, 276–281, 348–349
 Lown-Ganong-Levine pattern, 213, 344–345
 low-voltage limb leads, 202–203, 218–219, 244–245, 332–333
 low-voltage precordial leads, 68–69
 LPFB. *See* left posterior fascicular block

M
 MI. *See* myocardial infarction
 Mobitz type I second-degree atrioventricular block, 246–247, 254–255
 atrial tachycardia with, 254–255
 sinus bradycardia with, 300–301
 multifocal atrial tachycardia, 258–259
 myocardial infarction
 acute, 127
 acute anteroapical, 124–127
 anteroapical, 124–127
 anterolateral, 124–127
 anteroseptal, 218–219, 284–287
 inferior, 284–287
 inferior wall, 26–27, 40–41, 50–53, 138–139, 322–323
 lateral, 124–127, 258–259
 lateral wall, 146–147
 pseudo inferior wall, 134–135, 138–139, 306–307
 pseudo lateral wall, 146–147, 153

N
 narrow complex rhythm, 240–241
 narrow complex supraventricular tachycardia, 206–207, 212–213, 218–219, 222–223, 234–235, 238–239, 244–247, 250–251, 262–263, 268–269, 278–279, 292–295, 300–301, 304–305, 312–315, 342–343, 348–349
 narrow complex tachycardia, 189
 nonspecific ST-T wave abnormalities, 20–21, 26–27, 168–171, 202–203, 214–215, 254–255, 350–351
 no-RP tachycardia, 67, 73, 107, 206–207, 284–285

O
 orthodromic atrioventricular reentrant tachycardia, 162–163, 304–305, 307, 312–315

P
 P wave synchronous ventricular pacing, 180–181
 pacemaker-mediated tachycardia, 176–178
 pacing
 P wave synchronous ventricular, 180–181
 ventricular, 180–181
 poor R-wave progression, 218–219
 PR interval, 26–27
 precordial leads, low-voltage, 68–69

premature atrial complexes, 26–27, 46–47, 114–115, 162–163, 188–191, 294–295
 in bigeminal pattern with left bundle branch block, 12–13
 in bigeminal pattern with rate-related right bundle branch block, 2–3
 with increased preexcitation atrial bigeminy, 130–131
 with rate-related left bundle branch block, 6–7, 40–41
 with rate-related right bundle branch block, 2–3
 premature complexes, 200–201
 premature junctional complexes, 338–339
 premature ventricular complex, 34–35, 172–173, 287
 with compensatory pause, 22–23, 30–31
 prolonged atrioventricular conduction, 120–121, 168–173
 pseudo inferior wall myocardial infarction, 134–135, 138–139, 306–307
 pseudo lateral wall myocardial infarction, 146–147, 153
 PVC. *See* premature ventricular complex

Q
 QRS alternans, 57, 188–191, 304–305

R
 rate-related left anterior fascicular block, 22–23, 44–45
 rate-related left bundle branch block, 6–7, 34–35, 40–41, 162–163
 rate-related right bundle branch block, 2–3, 22–23, 30–31, 44–45, 47, 50–51, 62–63, 276–277, 338–339
 RBBB. *See* right bundle branch block
 right axis, 144–145
 deviation, 26–27, 50–51, 120–121, 276–277
 right bundle branch, 66–67, 84–85
 right bundle branch block, 86–87, 94–97, 100–103
 rate-related, 2–3, 22–23, 30–31, 44–45, 47, 50–51, 62–63, 276–277, 338–339
 wide complex tachycardia with, 92–93
 right ventricular conduction delay, 68–69, 262–263, 304–305, 326–327
 right ventricular hypertrophy, 258–259
 RR intervals, 185, 334–335
 R-wave progression, poor, 218–219

S
 second-degree atrioventricular block (Mobitz type I), 246–247, 254–255
 short PR interval, 26–27
 short PR tachycardia, 294–295, 314–315

short-RP tachycardia, 44–45, 50–51, 212–213, 292–293, 300–301, 312–313, 317, 342–343

sinoventricular rhythm, 201–203

sinus bradycardia, 26–27, 108–109, 250–251, 300–301, 344–345

sinus complexes, 13

sinus node reentry, 230–231

sinus rhythm

- with interpolated premature junctional complexes, 338–339
- normal, 2, 6, 12, 14, 20, 30, 34, 40, 46, 58, 68, 74, 80, 86, 96, 114–115, 120, 130, 134, 138, 146, 152, 156, 168, 188–190, 196, 208–209, 214, 286, 294, 306, 314, 318, 350

sinus tachycardia, 22–23, 38–39, 81, 124–127, 160–163, 170–173, 222–225, 228–229, 296–297

- with intraventricular conduction delay, 78–79

slow-fast atrioventricular nodal reentrant tachycardia, 67, 295

slow-slow atrioventricular nodal reentrant tachycardia, 51, 292–295, 313, 343, 345

ST-segment depression, 66–67, 304–305

ST-T wave abnormalities

- diffuse, 200–203
- left ventricular hypertrophy with, 322–323
- nonspecific, 20–21, 26–27, 168–171, 202–203, 214–215, 254–255, 350–351

supraventricular tachycardia, narrow complex, 206–207, 212–213, 218–219, 222–223, 234–235, 238–239, 244–247, 250–251, 262–263, 268–269, 278–279, 292–295, 300–301, 304–305, 312–315, 342–343, 348–349

sustained monomorphic ventricular tachycardia, 118–119

T

tachycardia

- accelerated idioventricular, 127
- antidromic atrioventricular reentrant, 144–145, 147, 163
- atrial. *See* atrial tachycardia
- atrioventricular nodal reentrant, 66–67, 72–73, 93, 101, 106–107, 162–163, 188–191, 206–207, 209, 284–285, 292–293
- Belhassen, 113
- endless loop, 177
- fascicular, 112–113
- junctional, 100–101
- left posterior fascicular, 113, 278–279
- long PR, 85
- long-RP, 84–85, 92–93, 218, 222–223, 234–235, 238–239, 244–247, 250–251, 268–269, 304–305, 348–349
- narrow complex, 189
- narrow complex supraventricular, 206–207, 212–213, 218–219, 222–223, 234–235, 238–239, 244–247, 250–251, 262–263, 268–269, 278–279, 292–295, 300–301, 304–305, 312–315, 342–343, 348–349
- no-RP, 67, 73, 107, 206–207, 284–285
- pacemaker-mediated, 176–178
- short PR, 294–295, 314–315
- short-RP, 44–45, 50–51, 212–213, 292–293, 300–301, 312–313, 317, 342–343
- sinus, 22–23, 38–39, 81, 126–127, 160–163, 172–173, 222–225, 234–235, 296–297
- supraventricular, 206–207
- ventricular, 112–113, 118–119, 121, 124–125
- verapamil-responsive ventricular, 113
- wide complex. *See* wide complex tachycardia

third-degree atrioventricular block, 334–335

3:1 atrioventricular block, 264–265

T-wave alternans, 188–191

T-wave inversions, 97

V

ventricular escape beat, 300–301

ventricular flutter, 277

ventricular pacing, 180–181

ventricular tachycardia, 112–113, 121, 124–125

- sustained monomorphic, 118–119

verapamil-responsive ventricular tachycardia, 113

W

wide complex rhythm, 194–195, 200–201

wide complex tachycardia, 84–85, 100–101, 112–113, 144–145

- atrial flutter with 1:1 conduction, 56–57
- atrioventricular nodal reentrant tachycardia, 66–67, 72–73, 106–107
- regular, 276–277
- right bundle branch block with, 92–93

Wolff-Parkinson-White (WPW) pattern, 130–131, 146–147, 152–154, 156–157, 162–163, 195, 306–307, 316–319, 343

- intermittent, 134–135, 138–139, 160–161

Wolff-Parkinson-White (WPW) syndrome, 131, 139, 145, 150–151

Notes